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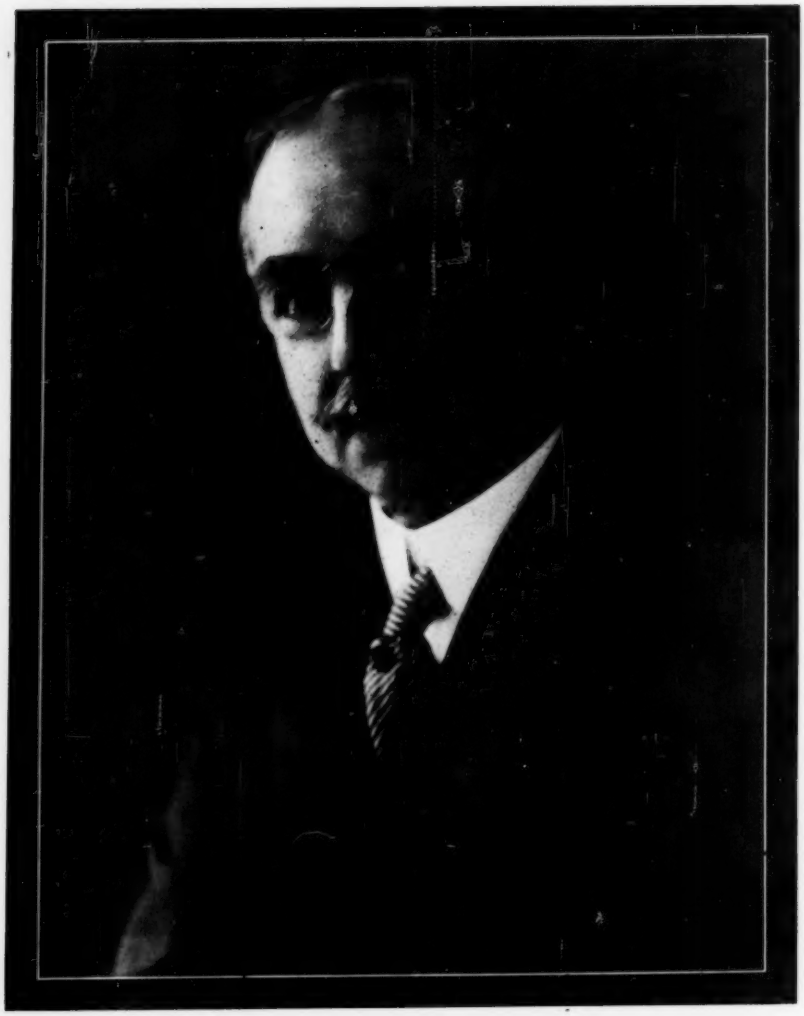

JAMES KELLY YOUNG, M.D.
1862-1923

VOL. VI, NO. 2 APRIL, 1924 Old Series: Vol. xxii, No. 2

The Journal of Bone & Joint Surgery

JAMES KELLY YOUNG, M.D.

Dr. James Kelly Young, an honored member of the American Orthopedic Association since 1890, died on August 28, 1923.

Dr. Young was born in Trenton, New Jersey, on April 29, 1862.

His father, William Young (born February 11, 1826), came to America from Staffordshire, England, at the age of eleven. He was joined later by his father, mother, and three brothers. The father, William Young, Sr., settled in Trenton, New Jersey, where, with his sons, he started a pottery business in the year 1853. It is noteworthy that it was this firm which made the first white wear of entirely American materials, all clays previously having been imported from England.

Dr. Young's mother was Ellen Eliza Kelly and her mother was Margaret Barrett, whose family in England was related to Elizabeth Barrett Browning.

Dr. Young was educated in the public schools of Trenton, graduated from the High School in 1879, and then attended the Medical Department of the University of Pennsylvania, graduating in 1883. In 1888 he studied surgery in Vienna. His professional career was begun in Philadelphia, and he was associated with Dr. William Porter, Dr. Sidney Roberts, and Dr. DeForest Willard. He held the positions of Associate Professor of Orthopædic Surgery at the University of Pennsylvania, Clinical Professor of Orthopædic Surgery at the Woman's Medical College, Consultant at the Woman's Medical College Hospital and Philadelphia Lying-In Charity,

Orthopædic Surgeon at the Philadelphia General Hospital, and Professor of Orthopædic Surgery at the University of Pennsylvania Graduate School of Medicine. He was the author of Manual and Atlas of Orthopædic Surgery (1894–1902); Synopsis of Human Anatomy, 6th Edition, 1889–1923. He was a contributor to Roberts' Orthopædic Surgery, Life and Writings of Dr. Sidney Roberts, to Keating's Encyclopædia of Children's Diseases and to International Clinics, besides being the author of numerous shorter contributions to medical publications.

Dr. Young had a large practice in orthopædic surgery and was noted for his success in his specialty and for his kind, sympathetic treatment of rich and poor alike. Especially with the poor was he sympathetic and helpful.

His health had been failing since 1918 on account of extra work during the World War, and in August, 1923, after an acute illness of about a week, he succumbed to valvular heart disease, with an acute endocarditis and complications.

Dr. Young is survived by his widow, Mary Wilson Young, whom he married in 1899, and by a son and a daughter.

NOTE ON A POSSIBLE SEQUELA AFTER OPERATION FOR THE REMOVAL OF THE INTERNAL SEMILUNAR CARTILAGE.*

BY NAUGHTON DUNN, BIRMINGHAM, ENGLAND.

We are all familiar with the painful neuromas following division of nerves in amputation stumps. I now wish to draw attention to the fact that the patellar branch of the internal saphenous nerve is divided by the usual incision made for removal of the internal meniscus of the knee joint.

As a rule, division of this nerve causes no untoward symptoms beyond a temporary loss of sensation in the infrapatellar region. In a small percentage of cases a painful neuroma may form in the scar and give rise to a sensation of pain as it passes over the margin of the condyle on flexion of the joint.

This condition first came under my notice in 1917, when an officer reported at the Military Orthopædic Hospital, Shepherd's Bush, that he had an occasional sudden sharp pain on the inner side of his knee. His history was as follows, and is typical of the condition I am describing:

"May 5, 1917. Lieut. S.

History. Internal semilunar cartilage right knee removed by operation four months ago. There has been no 'locking' of the knee since this, but he gets occasional sudden sharp pains referred to the inner side of the right knee.

Examination. No fluid in knee joint. Passive movements full and free. There is a definite thickening in the scar. This is tender to pressure, and the pain of which he complains is elicited when the thickening in the scar passes over the margin of the internal condyle.

May 10, 1917. Excision of scar—thickening of scar found to be a typical neuroma.

* Read before the meeting of the British Orthopædic Association in Birmingham on October 19, 1923.

June 11, 1917. Returned to duty—condition relieved.

Aug. 6, 1917. Re-admitted to hospital—complains that he is unable to ride his horse because of pain on inner side of right knee. Examination reveals a small, tender, mobile swelling in the subcutaneous tissue over the inner condyle of the right knee.

Aug. 10, 1917. Neuroma of patellar branch of internal saphenous excised and nerve shortened.

Aug. 30, 1917. Discharged well."

Since this I have had occasion to operate on seven cases where incomplete recovery after removal of the internal semilunar cartilage appeared to be due to involvement of this nerve in scar.

I recognise that it is a rare sequela, but I do not find any reference to it in the literature.

I show you the specimen of the nerve with its neuroma after removal.

The points I wish to draw your attention to are:

1. That a neuroma of the patellar branch of internal saphenous nerve may explain certain cases of incomplete recovery after removal of the internal semilunar cartilage.
2. That simple excision of the scar may not be sufficient to cure the condition.
3. That excision of the scar and evulsion of the nerve is the most satisfactory treatment.

The risk of this sequela may be obviated by retraction of the nerve or evulsion of the proximal portion of the nerve at the time of operation.

THE EPIPHYSES OF THE BONES OF THE EXTREMITIES AT PUBERTY.*

BY W. E. SULLIVAN, PH.D., F. D. GEIST, M.D., G. G. MUELLER, M.S.,
UNIVERSITY OF WISCONSIN, MADISON, WISCONSIN.

THE difficulty in finding a correlated description of all of the bones of the extremities at any given age first suggested the desirability of carrying out the present work. Good descriptions of individual bones may be found, but for the most part there is little sequence in recent work. It soon became apparent that the relation of the epiphyses to the joints was more desirable than a description of the epiphyses alone. If the work of von Brunn were more readily accessible this would be perhaps unnecessary.

Three cadavers of twelve to thirteen years were used. Rœntgenograms of all the specimens were made before dissection but the pictures were not enough better than those already published to warrant their use.

The work may be conveniently divided into the parts listed below:

A general discussion of epiphyses.
Vascularization of the epiphyses.
Epiphyses as a criterion of age.
Multiple ossification centers.
Summary of the relation of the epiphyses to the joints.
Description of the plates.

A GENERAL DISCUSSION OF EPIPHYSES.

While in this study we are treating primarily of the relation of the epiphyses to the joints, with a practical problem in mind, it is desirable to discuss briefly the epiphyses in a general way.

A comparative study of the epiphyses throughout the vertebrates is stimulating and has been carried out to good purpose especially by Parsons. In his "Observations on Traction Epiphyses" he writes:

* It has been found difficult to maintain our arrangement of the text and yet give full credit to all of the many workers who have preceded us in this field. The bibliography includes only papers read and used. Many excellent papers are omitted because they had no direct bearing on subject within the limits used.

Gordon Bemis, Irving Krohn and Howard Curl worked with us in the preparation and study of the material.

"In considering the subject of epiphyses I have come to the conclusion that there are at least three kinds.

The first are those which appear at the articular ends of long bones, whose shape corresponds more or less exactly with the cartilage covered area. These may be spoken of as pressure epiphyses, since they transmit the weight of the body from bone to bone.

The second kind form knoblike processes or, in some cases, caps over the tops of knobs where important muscles are attached to bones. Traction epiphyses will, perhaps, be a good name for these since they are connected with the pull of muscles.

The third kind includes those epiphyses which represent parts of the skeleton at one time of functional importance, but which, having lost their function, have now become fused with the neighbouring bones, and only appear as separate ossifications in early life. These I propose to speak of as atavistic epiphyses."

The tubercles of the humerus, the olecranon, the trochanters of the femur and the tibial tuberosity are examples of the structures discussed as traction epiphyses. The region of the symphysis pubis and the region of the tuberosity of the ischium, representing the epipubis of amphibians and reptiles and the hypischium of reptiles, are considered atavistic epiphyses.

It is difficult to be consistent and logical in any system of classification. This is true in the classification of epiphyses and there are obvious objections to the plan given. It may be argued that in the upper extremity of man weight does not enter into the problem. In traction epiphyses the pull may be such that the result is a pressure, and there would appear to be but little fundamental difference between the first two groups. In fact, a critical analysis shows no fundamental differences in the three groups except that it is reasonably clear that traction and atavistic epiphyses can be traced to independent skeletal elements. At present at least it is not possible to establish a similar condition for pressure epiphyses. On the whole, however, the value of the classification in correlating the structures in question far outweighs any objections that may be raised. Parsons seems fully cognizant of all of the faults of his plan and is surely to be commended for bringing a certain amount of order out of a chaotic condition.

While the relation of the epiphyses to a growing bone appears a simple one and we are now in a position to describe their method of development with a good deal of clearness and to enumerate with some degree of com-

pleteness a list of the vertebrates in which they are found, it is not possible to say why they appear and thus give their true significance.

Parsons suggests an explanation with a pathologic basis. He correlates the facts that ossification begins in the center of the cartilage and first in the larger cartilages with malnutrition at the center of the cartilage. This is in line with some experimental work in which ligation of the vessels is followed by calcification and ossification. The suggestion is an interesting one.

In reptiles, birds, and mammals, birds are conspicuous for the paucity of epiphyses. The recorded ones are a traction epiphysis at the proximal end of the tibiotarsus, an atavistic one at the distal end, and an atavistic one at the proximal end of the metatarsus. This paucity is suggestive in that birds have a relatively short period of growth. In pigeons adult size of the skeleton is attained in six weeks (Parsons). In the ostrich the rapidity of growth is phenomenal.

Epiphyses are especially well marked in vertebrates with a long period of growth. Parsons has pointed out that in some ungulates, ox and sheep, the epiphyses appear long before birth and that in a lamb of two days they are as well developed as in a man at twelve to thirteen years. This is what we would expect from a comparison of their life cycles.

From a survey of the hyaline cartilages in the sauropsida and mammals it appears that in general it is primarily an embryonic tissue and cannot maintain itself over a long period without ossification or at least calcification. Perhaps this should be modified to the extent of saying that it cannot maintain itself over a long period under either pressure or tension. Even the costal and laryngeal cartilages show relatively early indications of ossification. The ossification of the costal cartilages is peripheral and may proceed until a sheath is formed about the cartilage without its being extensively involved. It may be better to think of this as the formation of a membrane bone and to hold that the process is not comparable to epiphyseal ossification.

There are several well-known "laws" of epiphyses. One of the best known is that the epiphysis toward which the medullary canal is directed is the last to appear and the first to unite with the shaft. This holds fairly well for man, but the lower end of the fibula is an exception. Exceptions to this law are cited by Parsons for other animals, so it is best not to overemphasize it in referring to epiphyses in general.

So far as we have determined it is true that ossification first appears at the larger end of the bone, in the larger cartilaginous mass. This is again suggestive of the fact that a large epiphyseal cartilage cannot main-

tain itself over a long period without ossification and the larger the cartilage the shorter the period before the invasion by bone begins. In the carpus and tarsus, though there is not an absolute correlation between time and size, we do find that the capitate, hamate, calcaneus, and talus are the first to ossify. Further, if we take into consideration the bones having two or more centers there is no marked break in the time-size sequence. The fact that there is this time relation does not by any means solve the question of epiphyses but is another suggestive point.

Parsons has established a good case for the similarity of traction epiphyses to sesamoid bones. This, together with the atavistic epiphyses, would suggest that when the story of the skeleton is completely known it will develop that all of the epiphyses were at one time independent skeletal elements. This is the most attractive theory.

VASCULARIZATION OF THE EPIPHYSES.

It is not clear whether the epiphyses receive their blood through an extension of the vessels from the shaft or through an independent group of vessels. It has been suggested, but not demonstrated, that the vessels extend from the shaft to the epiphyses. We have examined a small series of a mammalian bone in which there was extensive ossification of the epiphysis and found the cartilage of the epiphyseal line intact. In a similar series of the tibiotarsus of the chick we found the cartilage of the epiphyseal line pierced by vessels. Pathologic data can be found to support either view.

EPIPHYSES AS A CRITERION OF AGE.

Practically all textbooks give the time data for the appearance and fusion of the epiphyses. We have omitted all definite statements in both cases. The collection of a considerable number of skeletons of known history by Professor Todd of Western Reserve University has made possible more accurate statements for the time of fusion. Data on this collection have been reported by Stevenson. The use of the X-ray by Pryor in attacking the problem has added new data on the beginning of ossification and has shown that it progresses the more rapidly in the female. Unpublished work by Hannon and Beffel of the University of Wisconsin strongly suggests that a correlation of ossification with sex, stature, and weight will give very accurate data for age. Until their data and additional data have been studied there is no point in including further time data.

MULTIPLE OSSIFICATION CENTERS.

In many instances more than one center of ossification is found. Throughout the description of the figures reference will be made to this condition. The best example of multiplicity of centers at this age is in the region of the acetabulum. Often isolated centers are found in the cartilage of the epiphyseal line. Throughout there is the suggestion that the epiphyseal centers are at first multiple, the number being progressively reduced by fusion, frequently to a single center. Sutural bones lend support to this suggestion.

SUMMARY OF THE RELATIONS OF THE EPIPHYSES TO THE JOINT CAVITIES.

In each instance the line of reflection of the synovial membrane has been used as the criterion of the relation of the epiphyses to the joint. Under any standard there are border-line cases where the line of reflection is on or near the cartilage of the epiphyseal line. In any doubtful case one should refer to the figures and make his own interpretation.

Clavicle. At the sternal end the upper part of the reflection of the synovial membrane is epiphyseal, the lower is diaphyseal.

Scapula. At the acromioclavicular and shoulder joints the reflections are epiphyseal.

Humerus. At the shoulder joint the lateral part of the reflection is epiphyseal, the medial is diaphyseal. At the distal end the anterior line of reflection is diaphyseal; the posterior line is diaphyseal in its medial part but epiphyseal in its lateral.

Ulna. At the proximal end the reflection is largely diaphyseal but around the tip of the olecranon is epiphyseal. At the distal end the reflection is epiphyseal for the radio-carpal joint but is practically on the epiphyseal line in the distal radio-ulnar joint, and the attachment of the capsule is diaphyseal.

Radius. At the proximal end the reflection is diaphyseal. At the distal end it is epiphyseal for the radio-carpal joint but diaphyseal for the radio-ulnar.

Metacarpal bones. The reflection on the first metacarpal bone is epiphyseal at the proximal end but diaphyseal at the distal. For the other metacarpals the reflection at the proximal end is necessarily diaphyseal. At the distal end it is epiphyseal but on or near the cartilage of the epiphyseal line.

Phalanges. At the proximal ends the reflection is epiphyseal, occa-

sionally approaching the epiphyseal line. At the distal ends it is necessarily diaphyseal.

Sacrum. At the auricular surface the synovial reflection is epiphyseal.

Os Coxæ. At the sacro-iliac joint the synovial reflection is epiphyseal. At the hip joint the reflection is epiphyseal.

Femur. At the hip joint the synovial reflection is diaphyseal. At the knee joint the reflection is epiphyseal; on the posterior surface of the bone the reflection is at the epiphyseal line. The capsular attachment on the anterior surface of the bone is definitely diaphyseal.

Tibia. At the knee joint the synovial reflection is epiphyseal. At the proximal tibio-fibular joint the reflection is epiphyseal. At the ankle joint the reflection is epiphyseal.

Fibula. At the proximal tibio-fibular joint the synovial reflection is epiphyseal. At the ankle joint the reflection is epiphyseal.

Metatarsals. The reflection on the first metatarsal bone is epiphyseal at the proximal end but diaphyseal at the distal end. For the other metatarsals the reflection at the proximal end is necessarily diaphyseal. At the distal end the reflection is epiphyseal.

Phalanges. At the proximal ends the reflection is epiphyseal. At the distal ends it is necessarily diaphyseal.

STERNOCLAVICULAR JOINT.

FIGURES 1 AND 2.

Figure 1 is a frontal section of the sternoclavicular joint.

The clavicular epiphysis has no center of ossification at this stage.

The reflection of the synovial membrane on the sternal end of the clavicle is epiphyseal in the upper part of the joint but diaphyseal in the lower.

The usual epiphysis in this region is the one on the sternal end of the clavicle. It does not appear until several years later.

As occasional centers there may be a small nodule on either side above the sternum for the suprasternal bones, which are sometimes found as separate bones.

The articular disc is present as usual and in this subject a meniscus is found projecting into the upper part of the cavity between the disc and the sternum. Into the lower part of the cavity between the disc and the clavicle there projects from the disc an incomplete labrum (not pictured). The presence in one joint of a complete disc, a meniscus, and

FIG. 1.

FIG. 3.

FIG. 2.

PLATE I.

The joint cavity is solid black, the cartilage is white, the bone shaded. The width of the joint cavity is exaggerated.

a labrum indicates quite clearly an attempt to maintain congruity between the opposed bones.

Figure 2 is a radiograph by A. W. Lindberg of the University Clinic. The subject was twenty-one years and five months of age, but is included that the relation of the clavicular epiphysis may be clearly pictured.

ACROMIOCLAVICULAR JOINT.

FIGURE 3.

Figure 3 is a horizontal section through the acromio-clavicular joint.

The epiphysis of the acromion, which is still in the cartilaginous stage, enters into the formation of the joint.

The line of reflection of the synovial membrane on the acromion is epiphyseal; on the clavicle it is necessarily diaphyseal, as there is no epiphysis at the lateral end of the clavicle.

At least four centers may appear in the acromion. Usually these soon fuse to form two centers.

An occasional acromial bone is found. Barnardeau, who considers it an atavistic structure, gives a very full discussion of its occurrence and significance.

This joint is very variable in structure, occurring at times as a synarthrosis and at times as a diarthrosis with a complete articular disc. The most common type is a diarthrosis with a rudimentary labrum. In this subject it was a simple diarthrosis with no suggestion of a disc or labrum but with a very thick cartilage on the clavicle. A comparative study of this joint in man would seem to establish amphiarthroses (symphyses, false synchondroses) as transitional joints between synarthroses and diarthroses.

SCAPULA AND SHOULDER JOINT.

FIGURES 4, 5, 6.

At the lateral angle of the scapula at this age there is a large amount of cartilage (Fig. 4). Ossification of the coracoid is well advanced and several small centers are present in the cartilage at its base. The most lateral of these is the subcoracoid which later extends into the upper part of the glenoid. Later another center appears in the glenoid. The acromial epiphysis is discussed with the acromioclavicular joint.

The inferior angle and the vertebral margin are entirely cartilaginous at this age.

Figure 5 is a frontal section of the shoulder joint. Figure 6 is a

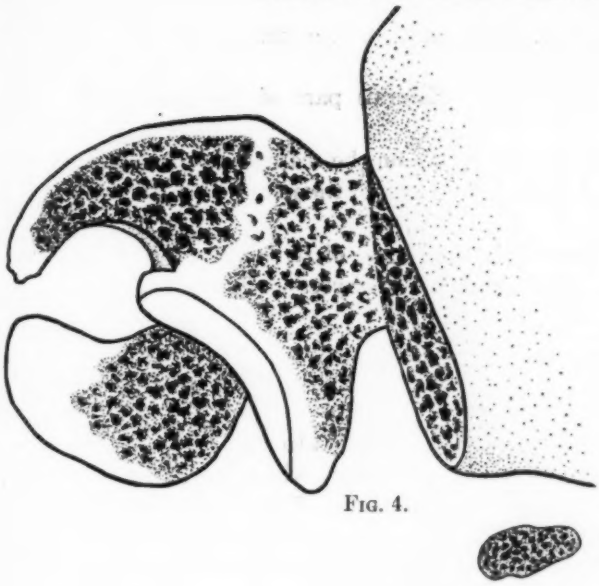

FIG. 4.

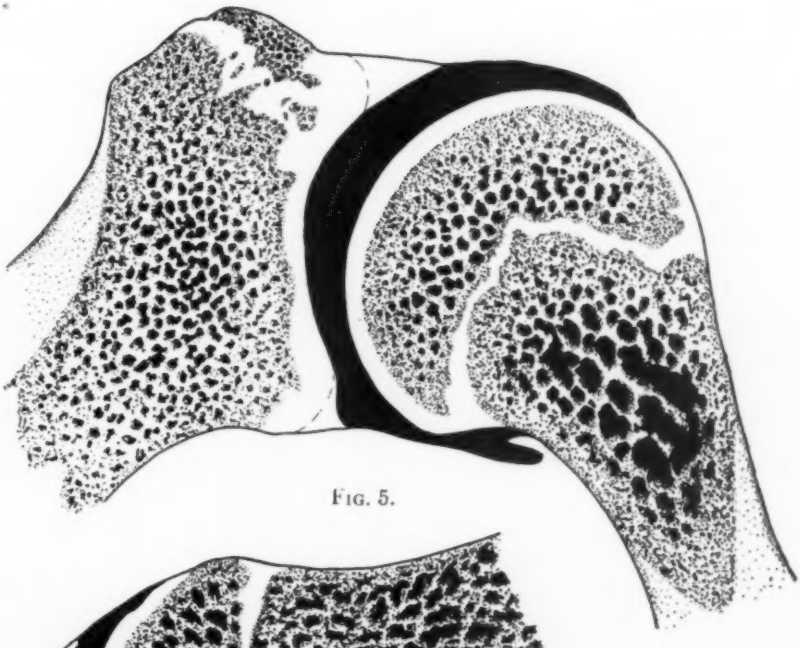

FIG. 5.

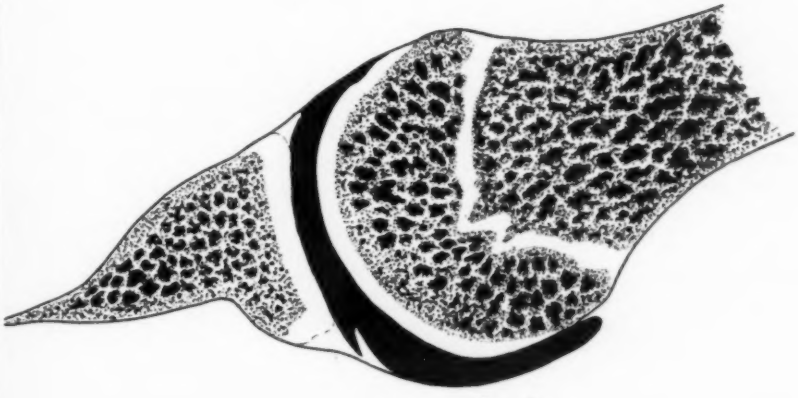

FIG. 6.

PLATE II.

The joint cavity is solid black, the cartilage is white, the bone shaded. The width of the joint cavity is exaggerated.

horizontal section through the cephalic (lateral) part of the joint with the arm abducted.

The reflection of the synovial membrane on the scapula is epiphyseal; on the humerus it is epiphyseal in the lateral part of the joint but diaphyseal in the medial.

The humeral epiphysis represents the fusion of the three centers for the head and two tubercles. The scapular epiphyses are discussed with Fig. 4.

THE REGION OF THE ELBOW.

FIGURES 7, 8, 9.

Figure 7 is the distal end of the humerus dissected to expose the centers of ossification in relation to the articular surfaces which are drawn in outline. The two epicondyles are extra-articular. The radial articular surface is largely on the center in the capitulum but extends onto the shaft. The ulnar articular cartilage is largely on the center in the shaft but also covers the small center in the trochlea and goes over on the center in the capitulum. There is an island of bone of good size between the centers in the trochlea and capitulum.

In the trochlear region there are three large islands of cartilage completely surrounded by diaphyseal bone. From the irregular nature of the epiphyseal line we would expect to meet this condition occasionally.

Figure 8 is a sagittal section through the trochlea and ulna. The humeral reflection of the synovial membrane is diaphyseal; the ulnar reflection is on the margin of the articular cartilage. It is epiphyseal around the tip of the olecranon but elsewhere is diaphyseal.

The two centers in the olecranon first noted by Fawcett are frequently described as one for the base and one for the tip. In the subject at hand one was lateral, the other medial. Toward the center they overlap, the lateral lying slightly anterior to the medial. One of these centers, probably the more lateral, may be present as a separate bone. Parsons has brought forth strong evidence to show that the olecranon is derived from a sesamoid bone.

The coronoid process of the ulna may have a separate center of ossification.

Figure 9 is a sagittal section through the radius and capitulum. On the humerus the anterior reflection of the synovial membrane is diaphyseal, the posterior is epiphyseal. On the radius the reflection of the synovial membrane is diaphyseal.

The tuberosity of the radius may have a separate center of ossification.

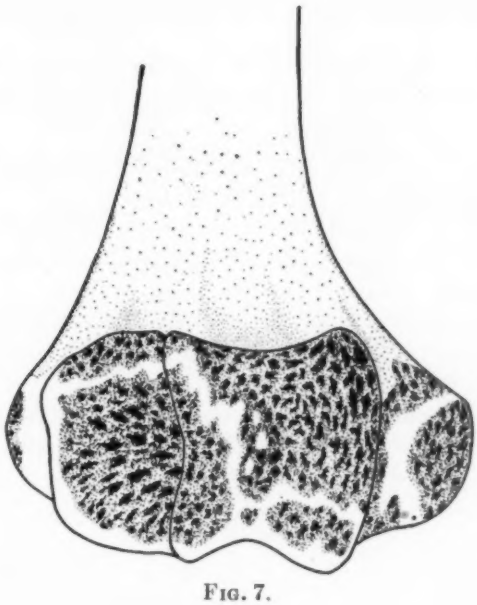

FIG. 7.

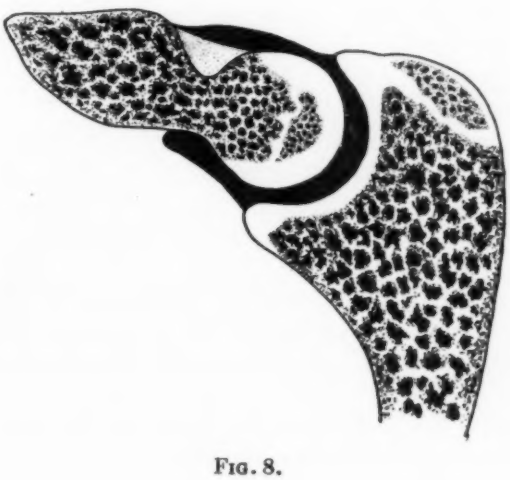

FIG. 8.

FIG. 9.

PLATE III.

The joint cavity is solid black, the cartilage is white, the bone shaded. The width of the joint cavity is exaggerated.

RADIO-CARPAL AND DISTAL RADIO-ULNAR JOINTS.

FIGURE 10.

Figure 10 is a frontal section through the bones of the forearm and proximal row of carpal bones.

The distal epiphyses of the radius and ulna show extensive ossification, as do the carpal bones.

The reflection of the synovial membrane in the radio-carpal joint is epiphyseal; in the distal radio-ulnar joint it is near the junction of the shafts and epiphyses.

In the subject at hand the cavities of the radio-carpal and radio-ulnar joints communicate freely with each other. The broken lines indicate the extent of the fibrous tissue.

The styloid processes of both radius and ulna may ossify from separate centers. Variations in the carpal bones are discussed with the hand.

THE HAND.

FIGURES 11 AND 12.

Figure 11 is a sagittal section through the first digit. The usual epiphyses at the proximal end of the first metacarpal bone and at the proximal end of the phalanges are found.

In the first carpometacarpal joint the reflection of the synovial membrane on the metacarpal bone is epiphyseal. In the metacarpophalangeal joint the reflection is diaphyseal on the metacarpal bone but epiphyseal on the phalanx. At the interphalangeal joint the reflection is diaphyseal for the proximal phalanx but epiphyseal for the distal.

Figure 12 is a sagittal section through the third digit. The epiphyses are present at the distal end of the metacarpal bone and at the proximal ends of the phalanges.

In the carpometacarpal joint the reflection of the synovial membrane on the metacarpal bone is diaphyseal.

In the metacarpophalangeal joint the reflection is epiphyseal for the metacarpal bone but is very close to the cartilage of the epiphyseal line. This is especially true on the volar surface. On the phalanx the reflection is epiphyseal.

In the interphalangeal joints the reflection is diaphyseal for the proximal bone but epiphyseal for the distal bone in each case.

A complete discussion of the variations of the bones of the hand is beyond the scope of this paper. They are well described in the papers of

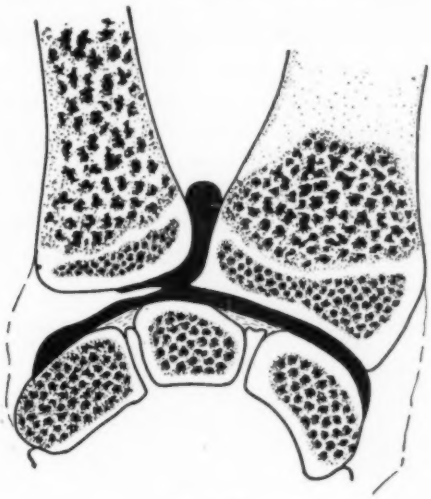

FIG. 10.

FIG. 11.

FIG. 12.

PLATE IV.

The joint cavity is solid black, the cartilage is white, the bone shaded. The width of the joint cavity is exaggerated.

Pfitzner and Thilenius which are adequately summarized in the anatomies of Quain and Rauber-Kopsch. Only the most important ones may be mentioned here. Two centers of ossification have been observed for the navicular, lunate, and triquetrum and a separate center for the hook of the hamate. The two centers for the navicular may remain separate. The os centrale is found as a separate bone in many animals, including the orang, and in man occurs as a separate cartilage. When present it lies between the navicular, lunate, lesser multangular, and capitate bones.

SACRO-ILIAC JOINT AND OS COXAE.

FIGURES 13 AND 14.

Figure 13 is a horizontal section through the sacro-iliac joint.

A small ossification center is present in the cartilage of the auricular part of the sacrum. This center can be considered to represent a beginning of the ossification of the epiphysis which subsequently forms for this part of the bone.

Ossification of the epiphysis for the auricular surface of the ilium has not commenced.

The line of reflection of the synovial membrane is on the cartilage for both the sacrum and the ilium; but on the ilium it is close to the junction of the cartilage and the bone.

Figure 14 illustrates the external surface of the os coxæ. The darker areas represent regions from which the superficial bone and part of the contiguous cartilage have been removed to demonstrate the lines of contact between the bone and the cartilage.

Two ossification centers are present in the posterior part of the cartilage of the iliac crest. One of these centers is independent, the other is continuous with the bone of the iliac ala. According to Fawcett the iliac crest ossifies from two centers—a ventral and a dorsal.

The center of ossification for the anterior inferior iliac spine has not appeared.

The inferior rami of the pubis and ischium have not fused. Two small ossicles are present in the lower part of the connecting cartilage. The centers for the epipubic, the ischial tuberosity, and the ischial spine have not appeared. The pubic tubercle may ossify as a separate center or as a part of the epipubic epiphysis.

The superficial part of the cartilage in the acetabular region was removed in thin slices. In the substance of the cartilage multiple, disseminated, irregular centers of ossification are indicated. The largest of

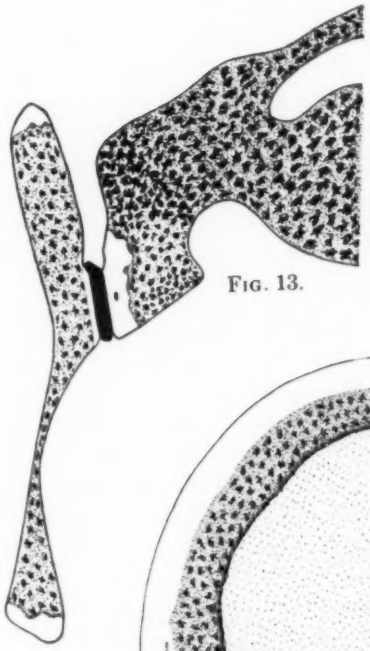

FIG. 13.

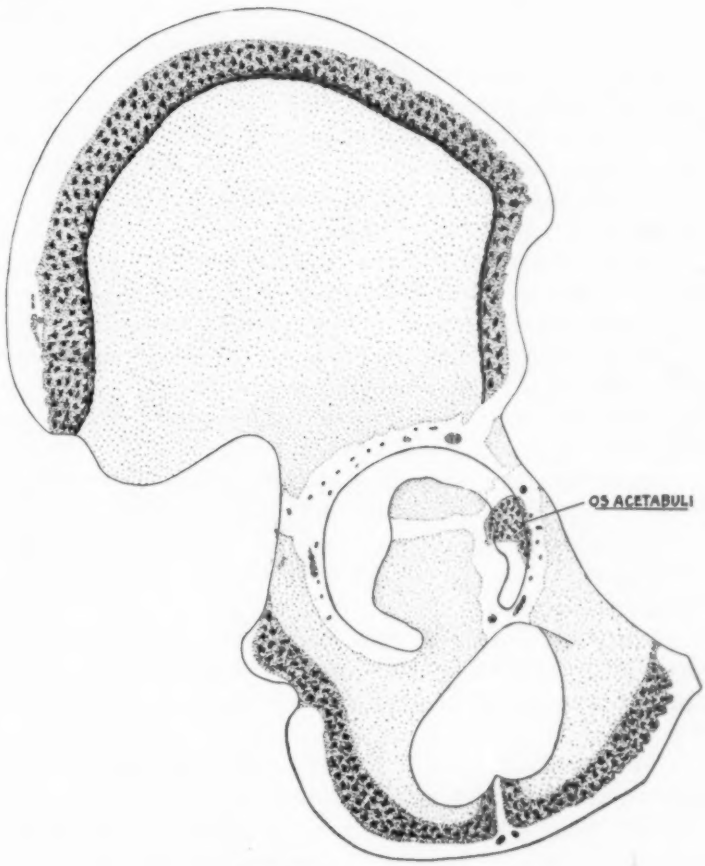

FIG. 14.

PLATE V.

The joint cavity is solid black, the cartilage is white, the bone shaded. The width of the joint cavity is exaggerated.

these is the os acetabuli. It occupies a position between the ilium, the pubis, and the ischium. The acetabular region of the os coxæ is an especially good example of the occurrence of multiple centers of ossification.

A definite ossified lamella for the iliac and ischial portions of the lunate surface of the acetabulum was not present.

OS COXAE AND HIP JOINT.

Figures 15, 16 and 17.

Figure 15 shows the acetabular region of the os coxæ in which all of the articular cartilage of the lunate surface has been removed. The outline of the lunate surface is represented to show its relation to the bone and cartilage. The position of the "os acetabuli" and its relation to the pubis, ilium, and ischium are more definitely shown than in the preceding figure. The os acetabuli forms approximately the upper half of the pubic portion of the acetabulum.

The position of the epiphyseal cartilage relative to the bone elements and to the acetabulum is indicated.

Figure 16 represents the internal surface of the os coxæ. The superficial bone and the superficial part of the intervening epiphyseal cartilage have been removed to expose the ossification centers in the cartilage.

The triradiate ossicle, a process of which projects between each two of the three chief elements of the os coxæ, represents the "os acetabuli." The triangular ossicle represented at the region of the ilio-pectineal eminence is also a part of the os acetabuli.

An independent ossification center is present in the cartilage between the ilium and the pubis; another between the ilium and the ischium.

The ilium and the ischium are connected by a small bar of bone.

Figure 17 is a frontal section of the hip joint passing through the ligamentum teres.

Numerous small centers of ossification are present in the cartilage between the ilium and the ischium. The largest of these centers, which is shown between the bone of the ilium and of the ischium, is part of the os acetabuli.

The ossification center for the head of the femur and that for the greater trochanter are well developed.

The synovial membrane is reflected on the glenoid labrum and on the neck of the femur. At birth the femoral reflection of the synovial membrane is on the cartilage of the neck.

The usual centers for the proximal end of the femur are: one for the

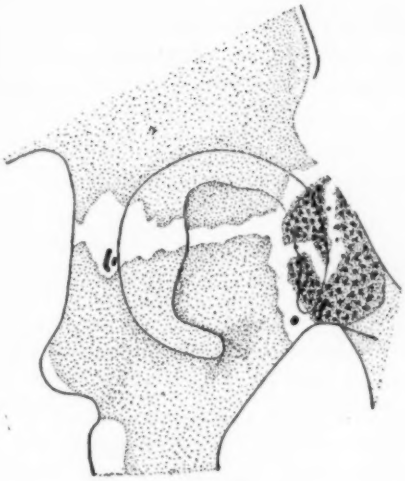

Fig. 15.

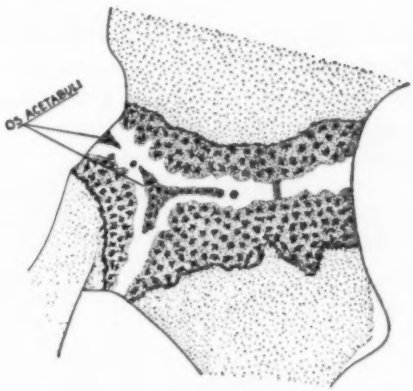

Fig. 16.

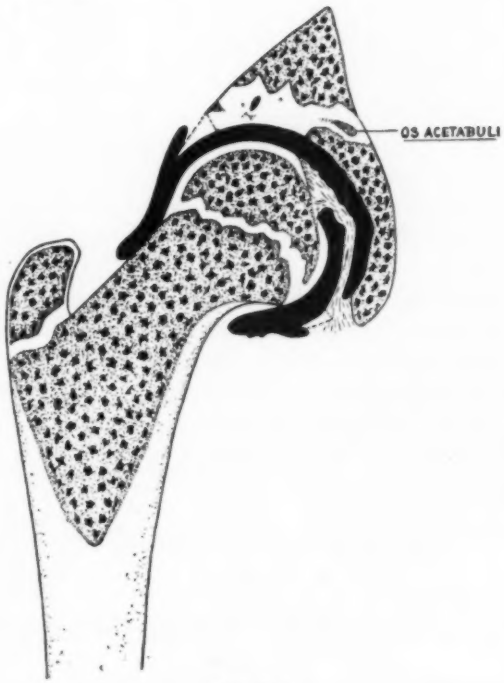

Fig. 17.

Plate VI.

The joint cavity is solid black, the cartilage is white, the bone shaded. The width of the joint cavity is exaggerated.

head, one for the greater trochanter, and one for the lesser trochanter. Bone formation in the greater trochanter may appear as a single center or as a group of granules which later coalesce. The center for the lesser trochanter does not show in the figure; in this specimen it had not yet fused with the rest of the bone.

The third trochanter occasionally ossifies as a separate center.

KNEE JOINT.

Figures 18 and 19.

Ossification of the patella and the epiphyses of the distal end of the femur and the proximal end of the tibia are well advanced.

Figure 18 is a sagittal section of the knee joint. The reflection of the synovial membrane from the anterior surface of the femur is epiphyseal. In the specimen from which the figure was taken, the suprapatellar bursa was continuous with the cavity of the knee joint. On the posterior surface of the femur the synovial reflection is at the epiphyseal line. In Fig. 19, a frontal section of the knee joint, the reflection of the synovial membrane from the femur is definitely epiphyseal. In both figures the tibial reflection of the membrane is epiphyseal.

In the section represented in Fig. 18 there is a synovial fold immediately above the fibro-cartilaginous meniscus.

The patella usually ossifies from a single center, but occasionally there are two lateral centers. Mouchet describes a case in which there were multiple centers.

The distal epiphysis of the femur and the proximal epiphysis of the tibia ossify from single centers. In the specimen from which Fig. 18 was taken the proximal epiphysis of the tibia and the tibial tuberosity are one continuous piece of bone. The tuberosity may ossify as an independent center or by extension of the bone in the proximal epiphysis. According to Kirchner, a secondary center may occur in the distal part of the tuberosity.

PROXIMAL TIBIO-FIBULAR JOINT.

Figure 20.

Figure 20 is a frontal section through the proximal tibio-fibular joint.

Ossification of the proximal epiphysis of the fibula is well advanced.

The reflection of the synovial membrane is epiphyseal on both the

FIG. 18.

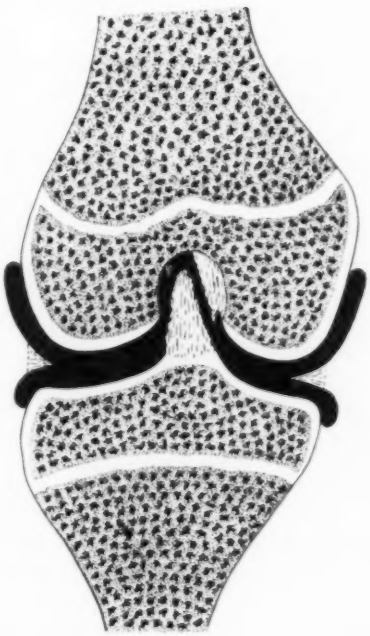

FIG. 19.

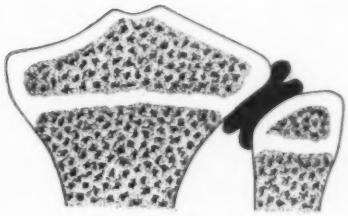

FIG. 20.

PLATE VII.

The joint cavity is solid black, the cartilage is white, the bone shaded. The width of the joint cavity is exaggerated.

tibia and the fibula. In the lower part of the joint it is reflected at the epiphyseal line on both bones.

A small meniscus is present in the lower part of the joint cavity. In the upper part of the joint there is a synovial fold.

ANKLE JOINT.

FIGURE 21.

Figure 21 is a frontal section through the ankle joint. The section includes the distal ends of the tibia and fibula, the talus, and the calcaneus.

Ossification of the talus, the calcaneus, and the distal epiphyses of the tibia and fibula are well advanced.

In the specimen represented in the figure, ossification of the distal epiphysis of the tibia had extended laterally and replaced the epiphyseal cartilage in this position. In a younger subject there would be a layer of epiphyseal cartilage on the lateral surface of this epiphysis.

There is no articular cartilage on the lower part of the diaphysis of the fibula in the inferior tibiofibular joint.

The synovial reflections of the ankle joint are epiphyseal both on the tibia and the fibula. A well developed meniscus is present with which the tibia and the talus articulate.

Ossification of the talus and the calcaneus will be considered in connection with Fig. 22.

The distal tibio-fibular joint is considered by some as a diarthrosis, by others as a synarthrosis. In consideration of the absence of articular cartilage on the fibula and the tibia where they come in contact, it is perhaps advisable to say that the cavity of the ankle joint is continuous with a bursa interposed between these two bones. Above this bursa the two bones are connected by fibrous tissue.

ANKLE REGION AND FOOT.

FIGURE 22.

Figure 22 is a sagittal section through the hallux and the ankle joint. The section passes through the calcaneus at the base of the sustentaculum tali and through the tuberosity.

Ossification of all of the bones included in the figure is well advanced. Epiphyses for the proximal end of the first metatarsal, the proximal end of the first phalanx, and the calcaneal tuberosity are present.

The reflections of the synovial membrane at the proximal ends of the

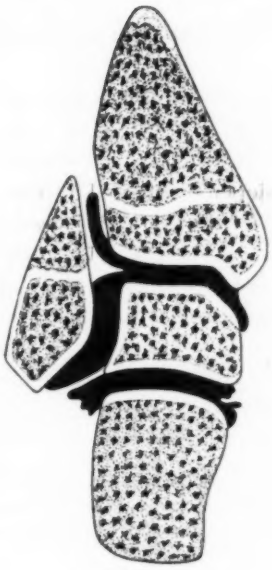

FIG. 21.

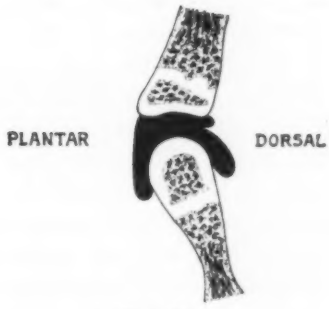

FIG. 23.

FIG. 22.

PLATE VIII.

The joint cavity is solid black, the cartilage is white, the bone shaded. The width of the joint cavity is exaggerated.

first metatarsal and the first phalanx are epiphyseal. Small menisci are present in most of the joints.

The epiphysis for the tuberosity of the calcaneus ossifies from several small centers which later fuse to form a single center before uniting with the body of the bone. The tuberosity of the navicular and the tuberosity of the base of the fifth metatarsal may ossify independently. The first cuneiform may ossify from two nuclei—a ventral and a dorsal. The second and third cuneiforms and the talus ossify from single centers. The posterior part of the talus may ossify as a separate center known as the "os trigonum." According to Hasselwander the calcaneus, cuboid, and navicular begin to ossify as two centers which soon coalesce. In the lateral four toes the epiphyses of the intermediate and distal phalanges were fused in one of the specimens, but only partially so in another. The epiphysis at the base of the distal phalanx of the hallux was fused.

The head of the first metatarsal occasionally ossifies from a separate center. When present it is intracapsular. Von Brunn describes its presence in specimens from individuals of 3, 6, and 9 years of age which he examined.

The medial sesamoid at the metatarso-phalangeal joint of the hallux may ossify from one to four centers. The lateral sesamoid usually ossifies from a single center, rarely from two centers.

A complete discussion of the variations of the bones of the foot is beyond the scope of this paper. They are well described in the papers of Pfitzner, Dwight, and Holland.

METATARSO-PHALANGEAL JOINTS.

FIGURE 23.

Figure 23 is a sagittal section through the metatarso-phalangeal joint of the fourth toe.

The synovial reflections are epiphyseal on both the metatarsal and the proximal phalanx.

A meniscus, incomplete on the plantar surface, is present in the joint cavity.

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THE OPERATIVE TREATMENT OF OLD CONGENITAL DISLOCATION OF THE HIP.

BY FRANK D. DICKSON, M.D., KANSAS CITY, MO.

An operation of election should never be performed unless very definite indications exist and there is a reasonable chance of securing improvement in the condition for which it is done. Any operation on an untreated or on an unsuccessfully treated congenital dislocation of the hip in an adult is an operation of election. It follows then that the indications for operative interference in such cases must be clean-cut and definite. In our own cases we have considered that only one indication existed, and this was disability of a degree sufficient to interfere with the ability of the individual to lead a normal life. There is, however, another factor which must be taken into consideration, and that is the desire to get rid of a disfiguring deformity. Whether the latter may be considered a legitimate indication I am unprepared at this time to state.

Given a definite indication, the next question is what degree of relief may be expected from operation? Does it offer enough promise to justify a rather serious procedure? We feel that it does, and present our results as a contribution to the solution of the problem of what to do with old congenital dislocation of the hip rather than as a solution.

As disability has been given as the indication for operation in these cases, let us first consider them as a whole with reference to disability. Eight cases of old congenital dislocation of the hip were examined, ranging in age from twenty-one to thirty-three years. Seven of the cases were unilateral, one was bilateral. The following were the symptoms complained of:

1. Pain in the lower back. This was present in all eight cases. In six cases it was severe enough to interfere with the patients' normal life, or to prevent them from following an occupation.

2. Pain in the dislocated hip. This was present in six of the unilateral cases, on very moderate exertion, as walking; in one, only when the hip was used considerably. It was not complained of in the bilateral case. In six of the eight cases the pain in the affected hip either prevented normal activity or interfered with occupation.

3. Shortening. This was found in all the seven unilateral cases and varied from one and one-fourth to three and one-half inches. One patient walked on crutches; three wore extension shoes. All felt that the shortening was a serious handicap, and ascribed much of their pain and lack of stamina to this.

4. Limp. This was present in the seven unilateral cases, and swaying gait was evident in the bilateral case. The limp was objected to but was not considered a disabling factor.

5. In all eight cases, the general endurance and capacity for work was far below par. This was due to pain on exertion, general tire on very slight exertion, and general lack of stamina.

To sum up then, we may say that the disability in these cases was due to three factors: a short extremity, an unstable hip joint, and postural defects, the result of the first two.

Since the cosmetic element has been mentioned, it might be well to state here that in one of our cases this was almost the only reason for operating, as the symptoms otherwise were not serious in character.

As to the results to be expected, we felt that if we could give a stable hip without any considerable limitation of motion and with a definite decrease in the amount of shortening, we would accomplish all that could be expected. We felt that if this were done we should secure an extremity that would be capable of bearing weight more efficiently and that this should relieve or at least decrease the back and hip pain, increase the endurance and render the individual more capable of effort. This we have been able in a large measure to do, and therefore feel that the procedure is successful.

Of the eight patients examined five were operated upon; all were unilateral. The bilateral case was not considered suitable for operation. Of the two unilateral patients not operated upon, one was leaving for China and we did not feel it desirable to undertake operation. The other case was postponed because of the general condition at the time of examination and the patient has not returned. Of the five patients operated upon, the results in four were satisfactory and the patients are leading normal lives. The fifth patient became infected, has marked limitation of motion in the hip and has derived no benefit from the operation. The results will be discussed in more detail in the individual case reports.

The procedure as carried out by us consisted in forming a new acetabulum by turning down a flap from the side of the ilium. No originality is claimed for the operation, as it is but an application of the procedure suggested by Albee some years ago and similar in some respects to that used by Ellis Jones for dislocation of the hip due to infantile paralysis.

The flap is taken from the side of the ilium just above the true acetabulum and includes that part of the ilium which forms the false acetabulum found in these cases. The flap turned down is about one and

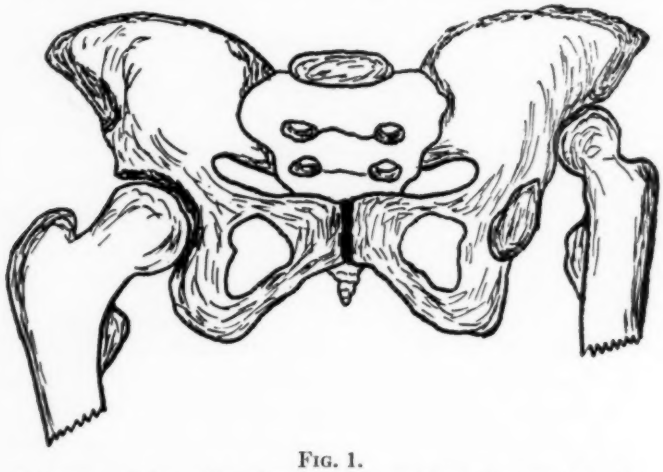

FIG. 1.
Diagram of the pelvis showing the position of congenital dislocation of the left hip.

one-half inches from base to tip, about one-half inch thick at the base, and broad enough to cover the entire head. The procedure in detail is as follows:

1. The patient was put on a Bradford frame, and traction applied for two weeks preceding the operation (Fig. 1).

2. For the operation a McKenna table was used. The patient was placed upon the table with the apparatus for making traction applied to both legs. In the first case adhesive plaster in the form of Buck's extension was used for making traction. In the second case ice-tongs, and in the last three cases a Steinmann's pin through the condyles was used for making traction on the dislocated hip.

3. The Smith-Petersen incision was used as it gives a good exposure, particularly of the area just above the true acetabulum. The capsule having been exposed through this incision, it was opened and freely cut

away in all directions. All structures which seemed to interfere with the downward movement of the dislocated head were divided, the hip being freely moved about to get a good exposure of all these structures. It was necessary in all cases to divide the ilio-psoas tendon, which formed a dense, firm, fibrous band and seemed to resist very distinctly the pulling down of the head. After the head had been freed, the adductors were divided subcutaneously. Traction was then applied to both legs and gradually increased, just sufficient traction being used on the sound side to prevent the patient slipping. As the traction was applied the patient's general condition and the circulation in the extremity were carefully watched to see that too much traction was not exerted. While the traction was being applied, the flap was turned down from the side of the ilium, a large, curved gouge being used for this purpose. In all cases, we succeeded in pulling the head down to a position about opposite the upper rim of the true acetabulum (Fig. 2). We found it practically impossible to get

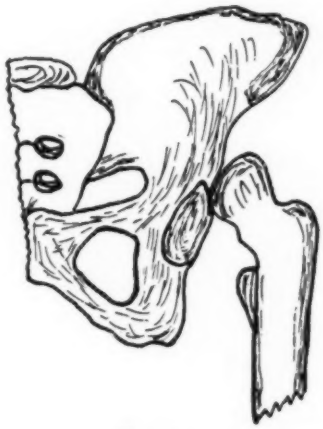

FIG. 2.

Diagrammatic representation of the position of the hip after traction for two weeks and after it has been pulled down by traction on McKenna table at the time of operation.

FIG. 3.

Showing process of turning the shelf of bone down from the ilium over the head of the femur.

it any lower without producing symptoms of shock. We therefore satisfied ourselves with this position and continued the turning down of the flap from the ilium until we had formed a new acetabulum with a good broad roof (Fig. 3). The flap turned down consisted largely, as stated, of the false

acetabulum, so that it was lined with a modified fibrous tissue which was smooth, thickened, and resembled somewhat normal capsular structure. We felt that this lining prevented in some degree the formation of adhesions and ankylosis. After the flap had been turned down until it was in apposition with the head of the femur, a wedge of bone was taken from the crest of the ilium and placed between the flap and the side of the ilium (Fig. 4). This wedge can be made to fit snugly and it is necessary to use only one or two catgut stitches to keep it in place. The wound was then closed

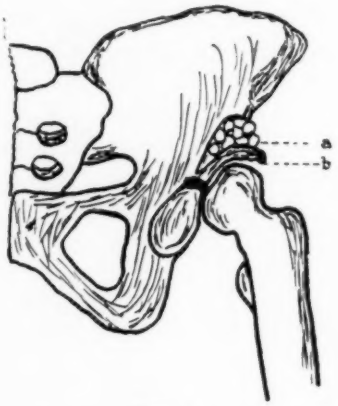

FIG. 4.

Diagrammatic representation of the hip after operation, showing the shelf, (b), which has been turned down over the head of the femur. (a) shows the wedge of bone which has been packed behind the shelf, (b), to hold it in place.

by layers and a plaster cast applied while traction was maintained. After the patient was returned to bed, an effort was made to keep some traction on the limb, in the first case by using the Buck's extension passed through slits in the cast, and in the other cases by leaving the ice-tongs or Steinmann's pin in situ and cutting out a window in the cast about it. I believe in the future that I will not use any cast at all, believing that traction will secure all the immobilization necessary, and without the cast this can be more advantageously used.

The cast was removed at the end of the fourth week, and daily massage and cautious movement of the hip commenced, the cast being replaced after the treatment. At the end of six weeks, traction was removed

and the patient was allowed up and about on crutches, with the cast on, and weight-bearing was gradually permitted. Traction, however, was reapplied when the patient returned to bed. Whether the traction had any important bearing on the result I am unable to say, but it was felt that traction should be maintained for some time for the purpose of keeping up the stretching of the structures in the hip, and to prevent if possible any tendency of the head to push up against the new acetabular roof. The cast was finally discarded by the end of eight to ten weeks.

The case reports are as follows:

Case 1, Miss ———, age twenty-eight years. Diagnosis: congenital dislocation of the left hip. Patient had had treatment as a child, but this was unsuccessful. She had worn an extension shoe since she was fourteen years old. Could walk about six to ten blocks and then became tired. Had pain in lower back when she exerted herself to any extent. Chief complaint was inability to be active or do what other girls her age did without great fatigue.

Examination showed that the left lower extremity was two and one-half inches shorter than the right. Motion was good in the hip, except limited abduction. Hip could be pushed up by making pressure against the sole of the foot, and on weight-bearing gave quite definitely. X-ray showed a good head and a well-marked false acetabulum.

Operation May 19, 1920. Operation as already described. Cast was removed on June 20, 1920, one month after operation. At this time there was three-quarters of an inch shortening; the hip seemed quite stable. Patient was allowed up on crutches at the end of six weeks and walked on crutches for another month, weight-bearing on left leg being gradually permitted. She returned home at the end of two months and was told to resume her normal life as rapidly as possible. From the time she commenced using her leg freely, which was about three months after operation, she complained no more of backache or tiring, and her ability to get about and do things progressively increased. At no time has there been any limitation of motion in the hip other than that she could not acutely flex the left hip joint. For all practical purposes, however, this limitation of flexion could be disregarded. As to her present condition, I will quote her own statement of April 15, 1923, made in a letter: "The new hip moves easily, with no pain. I have no pain in my back, and when the day closes my nerves are not on edge as they used to be before the operation. I drive, swim, and dance with ease, and feel confident that I can do anything anybody else can with my left leg. I am at present wear-

ing a half-inch elevation on my left heel." The patient was examined in May, 1923. At this time had three-quarters of an inch shortening, walked with a slight limp and the limitation of flexion was very slight.

Case 2, Miss B., age thirty-three years. Diagnosis: congenital dislocation of right hip. Patient had never had any surgical treatment, but her father was a physician, and he saw to it that the shortening was compensated for. She had worn a three-inch extension shoe for years. Had never had any marked disability; hip became tired only after considerable use, at which time she had pain in her lower back and some in the right hip. Could do almost anything that she cared to, but objected very seriously to wearing the extension shoe and was willing to do anything to get rid of it.

Examination showed the right leg two and one-half inches shorter than the left. Motion was good, except limitation of abduction and external rotation. X-ray showed a good head and a definite false acetabulum.

Operation, October 29, 1920. Operation as described. Cast was worn for four weeks and extension maintained for six weeks. Patient was then allowed up on crutches and gradually increased weight-bearing. She walked on crutches for six weeks after being out of bed, and then gradually resumed normal walking. Examination on March 26, 1921, showed that she had one inch shortening, all motion in the hip free, except she could not acutely flex the hip. This patient lives in California, and she has not been seen lately, but a letter from her on March 18, 1923, stated that she was then wearing one-fourth inch elevation on heel, had practically no limp, that she could swim, work in the garden, dance, and was carrying on quite an extensive horticultural enterprise at her home without tiring. To quote her own words: "I feel that the operation has been a complete success and the result has been a great satisfaction to me."

Case 3, Miss S., age twenty-eight years. Diagnosis: congenital dislocation of left hip. Condition had been undiagnosed. Patient had always had a limp. She came for treatment of severe back pain and pain down left leg and in the left hip.

Examination. The left leg was two and one-half inches short. The hip was quite movable and displaced upward very definitely on weight-bearing. A marked left lumbar curve was present. She walked with a distinct limp. Patient was treated for back pain for about a year, with high shoe, extension, and various other methods, without relief from the back pain. The possibilities of her being operated upon and making the

hip stable were explained to her and she at length insisted upon the operation being done, with complete recognition of the possibilities of failure.

Operation, November 8, 1920. The same procedure was carried out as described. On November 21st, patient ran a temperature of a 102° and there was drainage from the wound. This discharged for three weeks. Extension in her case was maintained for six weeks and cast retained about the same time. At the end of six weeks she was allowed up on crutches which she used for six weeks more and then was allowed to discard them. We felt in this case that we had much more limitation of motion than in any of the other cases. Every effort was made to increase this, but with very little success. On November 19, 1921, the hip was stable, pains in the back were very little improved. There was considerable limitation of motion of the hip in all directions, although there was enough motion for her to sit and to walk without much discomfort. The shortening was one inch. This patient has been seen from time to time, has had practically no increase in the amount of motion in the hip and only occasional relief from the backache. In this case the patient has derived no advantage whatever from the operation other than that she has a stable hip. This has not improved her back pain or her general condition to any extent.

Case 4, Mr. L., age twenty-four years. Diagnosis: old pathologic dislocation of the hip. When he was twelve years old, patient had an infection in the left femur, which was operated upon and pus evacuated. This discharged for over a year, many pieces of bone coming away. Had many operations for abscesses in various parts of the body, too numerous for the patient to attempt to describe. Had had no discharge from the hip for twelve years. When seen at first by us, the patient walked with a marked limp and most of the time had to use crutches. The hip did not pain him except when weight was borne on it, at which time it became very painful. He also had pain in the back, and in order to follow any occupation he had to use crutches.

Examination of right leg revealed much atrophy and shortening of extremity, with several scars of old incisions about the hip joint. The right lower extremity was three and one-half inches shorter than the left. Adduction and flexion of the hip joint were good. Abduction and rotation were almost absent. The tip of the great trochanter was about on a level with the anterior-superior spine of the ilium. There was marked adduction deformity and marked tilting down of the pelvis on the right side. Spinal curvature was present, chiefly in the lumbar region. X-ray showed

that the head was partly destroyed but fairly robust, and was dislocated upward and backward.

Operation in February, 1921. Operation as described. Cast was kept on for four weeks, extension for six weeks. Patient was then allowed up on crutches, with gradual weight-bearing. Examination on May 7, 1921, showed that patient was walking quite well with cane. There was some limitation of motion still present. In December, 1921, was examined again. Patient was then walking with some limp. Motion in the hip was good. Flexion was slightly limited, but abduction and rotation, which patient had to only a limited degree before, were present almost in a normal degree, shortening one and three-quarters inches. He walked about, using a cane, and for the past eight months had been following the occupation of salesman through the country towns of Kansas. A letter received from this patient May 17, 1923, states that he is now living on a farm in Oklahoma, and he gives the following statement as to his present condition. "I am feeling fine, weigh one hundred and fifty-four pounds and have not had any trouble whatever with my hip. Most of my work here is outside and I do a great deal of walking without pain or getting tired. I use a cane out-of-doors, but think I would be better if I did not carry it. I also want to say that my hip is practically as limber as my other one. I am more than pleased about the outcome of my operation."

Case 5, Mr. T., age twenty-one years. Diagnosis: congenital dislocation of left hip. Patient had never had condition diagnosed and no treatment had been given. He walked with a limp, tired very easily, and had pain in back and left leg. As a result, he was unable to work his farm and came to see whether he could be put in condition to do farm work.

Examination showed left leg one and one-quarter inch shorter than the right. Hip movements were normal, except for limited abduction. The hip gave on weight-bearing, but otherwise did not seem particularly movable in examination. Inasmuch as this man was practically unable to work his farm, an operation was advised.

Operation in September, 1921. Operation as described. Cast was removed in four weeks, but extension was maintained for six weeks. The patient was allowed up at the end of this time on crutches, as in the other cases. Examination in February, 1922, showed motion practically normal, flexion being very slightly limited. Left leg was about three-quarters of an inch short. Hip was stable, patient had no pain and could work all day. He was examined April 8, 1923, and aside from the fact that he

still has some limp his condition is practically normal. He states he is working about ten hours a day on the farm, without any discomfort whatsoever.

To summarize the result of these five cases: four, or eighty per cent, have a result which is quite satisfactory; there is an absolute failure in one case, or twenty per cent. We feel that in the case which was a failure the failure was due to infection and not to the operation. Admitting that infection is probably a certain cause of failure, it does not seem that the possibility of its occurrence is a contraindication to operative interference in these cases.

As in none of the five cases were we able to get the affected extremity equal in length to that of the good side, I am inclined to feel that the benefit which the four successful cases secured was due more to stabilizing the hip joint than to the increase in length secured. In fact, on examining the late roentgenograms of these hips, I have been impressed with the apparently high position of the head; three of the five cases have been measured within six weeks and the shortening is the same as that shown by the measurements taken within six weeks of the operation, so there has not been any upward displacement since weight-bearing has been constant. I am inclined to believe that as so frequently happens in the roentgenograms of congenital dislocation of the hip, the picture we get is not a true index to the actual condition present.

The only limitation of motion which has occurred has been inability to acutely flex the hip and this is comparatively unimportant, as it is a movement which is only occasionally used. Its lack in no way interferes with ordinary activity.

While we appreciate that five is perhaps a small number of cases on which to base conclusions, still the results have been satisfactory enough to seem worth while reporting. Basing our conclusions on these five cases we feel that the procedure is useful and worthy of consideration in cases of congenital dislocation of the hip in adults where there is sufficient instability to interfere with the individual's ability to lead a normal life.

DISCUSSION ON PAPER OF DR. DICKSON.

Dr. A. Bruce Gill, Philadelphia, Pa.: Dr. Dickson's communication opens up for our consideration a large, important, and almost virgin field of orthopædic surgery. For many years orthopædic surgeons have been endeavoring to perfect methods of reduction of congenital dislocation of the hip in children; first by manipulation, then by open operation, and again by manipulation. As we know, some surgeons have returned again to the open method in all, or almost all, cases.

The entire question is still *sub judice*. The bloodless method, however, has been followed by most surgeons in recent years, and statistics have been published which show failure to reduce or to maintain satisfactory reduction in a fairly large percentage of cases, possibly forty to fifty per cent. all told.

Another fairly large group of patients, who have passed an age limit, fixed by different surgeons from eight to twelve years, have been pronounced hopeless and have been denied operation by any method.

In these two groups, therefore, there must be more than half of the total number of cases of congenital dislocation of the hip. What has become of them? If they have been able to lead normal lives with unreduced hips, why should we greatly concern ourselves with the question of reduction at any age?

During the past four years I have operated upon nineteen patients between the ages of twelve and twenty-four years. Some of them had been operated on by bloodless and by open methods, others had not. The older cases came to my clinic because they were having pain, so great in some instances as to produce complete incapacity to walk more than a few steps or to stand more than a short time on the feet. My experience thus corresponds with Dr. Dickson's.

The kind of operation which I have employed has varied with different types of cases and with the difficulties to be overcome in individual cases. Some patients have fairly well formed false acetabula, but suffer from pain and limitation of motion. Pain has been entirely relieved and mobility increased by deepening the false acetabulum and allowing the head to remain in it. This does not, however, correct the lordosis and restore normal gait. But in some of my cases it has been eminently satisfactory. Again, when the false acetabulum has been shallow and the patient suffered from pain and instability, a bone flap has been turned downward and forward above the head without opening the capsule or disturbing the position of the head.

In the majority of cases there has been no false acetabulum present. The operation employed is then as follows: Through the Smith-Petersen incision the tensor fascia femoris and the anterior part of the gluteus medius are separated subperiosteally from the pelvis and turned downward. The anterior part of the incision is carried downward along the anterior margin of the tensor femoris until we come to the anterior inferior spine of the ilium. The capsule is then detached from the ilium from below the inferior spine backward. The finger can then be inserted within the capsular cavity and the head of the femur and the acetabulum palpated. In the older cases the acetabulum almost always is found to be obliterated completely. A longitudinal incision is then made through the capsule in order to disengage the head and bring it into view. The thigh is then flexed and abducted, and it is observed how far downward and forward the head may be carried. It usually comes with ease to a position just below and behind the inferior spine. An excavation is then dug in the pelvis, begun at the posterior upper margin of the acetabulum and enlarged backward and upward until the head can be placed in it without much tension. On one occasion in a twenty-four year old girl I made the hole at the site of the original acetabulum and forced the head into it with great effort, tearing off the cartilage of the head in the attempt. This I believe to be a mistake, as it leads to ankylosis. The excavation is made deep enough to hold the head in this position when the thigh is extended and abducted moderately. A bone flap is then turned downward and forward above and behind the head, and

the pieces of bone which have been removed in making the acetabular cavity are packed in behind and above the flap to keep it in position. Other pieces of bone for the same purpose may be removed from the side of the pelvis, if necessary. Sometimes the flap of bone has broken off at the pedicle. In such instances I have found that it can be driven perpendicularly into the side of the pelvis with a small hammer. No sutures are placed in the capsule as the free edges fall of themselves into place about the bone flap as the gluteus medius and the tensor femoris are replaced and sutured to the fascia along the posterior crest of the ilium. The extremity is kept in a plaster cast for six weeks in extension and slight abduction, a good walking position, should there be much limitation of motion. At the end of six weeks the cast is removed, and massage, baking, active and passive motion are begun. Weight-bearing is not allowed for twelve weeks.

I do not wish at this time to make a complete report of these nineteen cases as insufficient time has elapsed after operation on a number of them. Stability we may always be sure of obtaining. To secure mobility is more difficult. Certain mistakes in my earlier operations, such as forcible leverage of the head into the acetabulum, separation of the cartilage of the head, the digging of too deep an acetabulum, retention in plaster for too long a time, lead in some instances to fibrous ankylosis. These things, together with much other interesting data, will be presented at a later time.

One other consideration is deserving of great emphasis. Why should we permit these individuals with dislocated hips to pass beyond childhood without efficient reduction? If we cannot secure a good result by bloodless methods, I am strongly of the opinion that we should resort to open operation. And the lessons which I have learned from the surgery on the older cases I have more recently been applying to the younger ones with apparently the best of results. Open operation on these children has invariably shown why, because of horn-glass capsule or obliterated acetabulum, closed reduction was unsuccessful and impossible.

Dr. Robert B. Osgood, Boston, Mass.: I would like to ask Dr. Gill to outline the technique of his operation.

Dr. Fred Albee, New York City: A considerable number of these cases have come under my observation during the past ten years; in fact, two cases that proved very successful were patients of Dr. Jones of Los Angeles, and I operated on them for paralytic dislocation at Dr. Jones' Clinic in 1915.

I believe we should divide these cases into two classes: namely, the cases where the hip will not stay in after being put in (in which case the problem is to increase the depth of the acetabulum); and those cases in which the child has become so old or the femoral head is riding so high that reduction is impossible, or if it is possible, the resulting trauma to the femoral head or the acetabulum would be too severe. I agree absolutely with Dr. Gill that in the difficult cases with much shortening we should not be too anxious to get the hip into the acetabulum. It is well known that if we traumatize the head of the femur or even the acetabulum too much during reduction, the trauma will produce a limitation of motion in that hip. It is best in selected cases to aim for a less ideal result and to build the acetabulum just above the true acetabulum rather than try to place the femoral head in the acetabulum.

The conditions that we find at the hip from the surgical standpoint vary, and therefore our plan of operation and technique should vary in accordance with the conditions actually found after the hip is laid open. When approaching the hip joint by the Smith-Petersen method, one can turn down a generous-sized graft from the outer table of the ilium. If one turns down the bone and produces a shelf above the capsule at the border of the acetabulum, or above it, the hip may be very satisfactorily stabilized, although the radiogram does not show well the features of the bony reconstruction. X-ray demonstrations, and especially lantern slides therefrom, do not therefore satisfactorily demonstrate as a rule the results in these cases. I believe that is the reason that some of the lantern slides shown by Dr. Dickson do not show his very satisfactory results so well. I believe this work is very important and that it should be studied and tried by every member of the association —certainly everyone should take under advisement operative treatment of the many cases of congenital or paralytic dislocation, in which the hip will not stay in the acetabulum after "bloodless" reduction.

Dr. John Ridlon, Chicago, Ill.: I rise simply to have written into this record the fact that Alexander Hugh Ferguson at least twenty years ago stabilized a congenital dislocation of the hip by turning down a flap from the ilium. He might have reduced the dislocation as there was only one inch of shortening. But he simply made an incision through the outer surface of the ilium above the head where it lay on the ilium and turned that down as a shelf over the head. He did this when the child was two and one-half years old. Six years later the patient came to me and asked for a reduction. The head had remained just exactly where it lay when the operation was done. There was the same inch of shortening and there was the same normal range of motion that a congenital hip should have. It stood as a perfect operation for what was done. The place in the ilium where this semicircle was taken out and turned down was easily seen in the radiogram. I think Ferguson should have the credit of doing the first work toward this very valuable work that is now being done.

Dr. Gill has spoken of relapsed cases. Of course we all have relapsed cases. It is a question whether certain ones should have a cutting operation and others should not. Certainly all those Dr. Dickson reported with pain and suffering should have a cutting operation, though I am surprised that one should have so many in a short time. I have done a great many congenital hip operations and I have had my full share of relapsed cases, but I have had no relapsed cases disabled as these cases that were reported and none of them suffered pain, their disability simply being due to the shortening of the leg. Only two patients have I ever seen with disability such as has been described. One was in a woman past fifty, a bilateral dislocation. She weighed two hundred and thirty pounds, and never having had any operation at all she was disabled for walking more than a couple of hundred feet, in my opinion because of her very great weight. The other, also a bilateral dislocation—one hip that was up and the other up only a little way—had never had any attempts at reduction or treatment whatever, and she had pain in the hip that was dislocated far up. Those are the only two out of all my cases that suffered as these patients had suffered, but patients who are unable to walk should have an open operation.

Dr. J. O. Wallace, Pittsburgh, Pa.: I have seen a number of Dr. Gill's patients in the last four years and just last Monday I spent a day with him in Philadelphia and saw quite a number of cases and saw what he considered his worst results. To my mind they were all improved. To a patient with a unilateral congenital dislocation who has pain and a riding up of the head, even a stiff hip in a good position I think is preferable to the former condition. Two or three of his cases which had considerable stiffness were abducted but I believe in the course of the next year those patients ought to get very good motion.

I have done a number of these cases. I remember three total cases and seven unilateral dislocations. Now I have turned down flaps; I have also taken a wedge from the crest of the ilium. With the Smith-Petersen incision it is a very simple matter to take quite a layer of bone from the crest of the ilium and put on the acetabulum. X-ray pictures taken after operation make you feel as though you did not do much good. You think the hips are still way up, but yet it is the functional result that counts every time, whether the patients can walk around or whether they have a waddle like the majority of patients with double dislocation.

Dr. A. F. Moxey, Philadelphia, Pa.: The subject now under discussion is one of practical importance to every orthopaedic surgeon. A new field is being entered in the treatment by operative means of these old cases of congenital dislocation of the hip. Dr. Dickson's paper is a very able presentation of a subject that is of importance to us, a subject that should be carefully studied and thought out.

The question might be asked, How early does a congenital dislocation of the hip become old? "Age" here is used more as an index to the ease of reduction than to the physical age of the patient. My observation has been that it becomes old, like some other dislocations not of congenital origin, rather soon—that is, at a comparatively early age and not necessarily when adult life is reached. We all have had cases, no doubt, which we could class as "old" congenital dislocations of the hip in boys and girls, mostly girls, long years before they reached adult life. Such cases might very well be included in the same classification as "adult" cases and be discussed with propriety here. Radiograms of some of these have been shown on the screen by Dr. Gill.

I disagree with Dr. Ridlon as to the complaints that bring these older patients to us. They come to us as they grow older and as the body-weight becomes greater because of the increasing pain and discomfort on standing and walking. Increasing body-weight produces a greater degree of strain on the soft tissue about the dislocated hip, with consequent greater pain and discomfort to the patient. We operate to give relief. In operating we either replace the femoral head in the acetabulum, or make a new acetabulum for it, so that the weight-bearing strain is taken up, as it should be, by bone and not by soft tissues. When this normal relationship is restored, symptomatic relief and freedom from pain are obtained.

In Philadelphia Dr. Gill and I have also operated upon various types of pathologic dislocation of the hip, and there is very little difference between these and the congenital dislocations in the indications for and type of operation performed.

Before resorting to open operation in these older cases of congenital dislocation of the hip, bloodless reduction is usually first attempted, up to the age of twelve or sixteen years. This failing, or in the event of the case relapsing, open

operation is then performed. One of the difficulties following the closed method of reduction has been the disability ensuing from limitation of motion. That has been the common experience of every orthopædic surgeon. One of the mistakes made in our earlier operative cases was too long immobilization in plaster after operation. We have not immobilized our later cases as long as we did our earlier ones. Six weeks in plaster is long enough, at the end of which time the cast is removed and passive motion started.

Another mistake made in our earlier operative cases was the production of too much traumatism at the time of operation. We do not use as much traction and force at the time of operating as we did at first. We do not use the hip-spoon, like a shoe-horn, any longer for prying the head down into the acetabulum. I deem it better to enlarge the acetabulum upward if necessary, thus placing it at a higher level. Or a new acetabulum is made by gouging out the ilium, usually at that point, well forward, opposite which the femoral head can be placed without making excessive traction on the leg. We have denuded the femoral head of its articular cartilage in at least one of our cases in an unwise attempt to reduce the head into the original acetabulum, using too much force in the attempt, and this patient failed to get motion in the hip joint and had a poor result functionally because of the ankylosis.

Another thing: we do not put our cases up in plaster in as wide abduction as is done after bloodless reduction. We have had difficulty, as you all doubtless have had, in correcting the abduction in some of our cases when the casts are removed after bloodless reductions. That is due to too long immobilization in extreme abduction. We had this same experience, only to a greater degree, with some of our earlier operative cases that we placed in plaster in either the frog-leg position or in extreme abduction. Now we no longer place our operative cases in plaster casts in the frog-leg position, but in casts, pelvis to toes, affected leg only, at 30° abduction (or sufficient to hold the head in its newly formed socket); and this cast is removed at the end of six weeks, when massage and passive motion are begun. If a bone flap has been turned down from the ilium above the femoral head, so as to give a wider weight-bearing upper margin to the acetabulum, a Buck's extension is applied to the leg after removal of the cast so as to prevent upward pressure of the femur disturbing this newly formed upper acetabular margin until it solidifies and becomes firm enough to withstand such pressure. Walking and weight-bearing are begun cautiously, with crutches at first, and usually not before twelve weeks have elapsed from the time of the operation.

Again, I repeat, this subject is both an extremely interesting and a very important one, and Dr. Dickson deserves great credit for the admirable manner in which he has called it to our attention.

Dr. John L. Porter, Chicago, Ill.: I would like to ask two questions: first, I want to ask Dr. Dickson, if he abandons the plaster cast, what is he going to do during that period between the time he finishes the operation and the time the patient is put to bed? When the patient is picked up by the orderly or by the interne and moved from the table to the cart and from the cart to the room and to bed, there is a period during which a great deal of traumatism may take place as far as the new flap is concerned, a period that is critical, though it may last only fifteen minutes, so far as the final result is concerned.

I would like to ask Dr. Gill and Dr. Dickson what they have observed in the histological condition of the head. Does not considerable atrophy take place in some of those cases after weight-bearing falls on the replaced head? I ask that because I have replaced by closed reduction and ordinary manipulation a head in a girl 15 years of age. The reduction was comparatively easy. The patient was put to bed for three weeks with 20 pounds traction. I was very fortunate in having audience when I did that reduction. Dr. Ryerson stood beside the table and saw the head replaced in the acetabulum. It went in very distinctly and sharply. I patted myself on the back and thought I had accomplished something but I wished that I had never reduced it, because the head was so atrophic and the circulation was so poor that it underwent absorption and destruction, and the motion resulting from that reduction was much less than the motion before reduction. I am curious to know if the same thing does not take place after the head is placed in an artificial bony acetabulum.

Dr. Frank D. Dickson, Kansas City, Mo. (closing the discussion): I first want to thank Dr. Ridlon for calling my attention to Dr. Ferguson's early procedure. I must say I was not aware of that fact.

As to Dr. Ridlon's contention that these patients do not suffer pain, I think he is decidedly wrong. Three of these patients did not know they had a congenital dislocation of the hip. They came because they had pain in the back and leg. I did not say they could not walk, but they could not walk to any great extent and they could not follow their occupations. In the West there are a great many cases that have never had treatment. These cases came from as far west as California and as far east as my own state of Missouri. They all came individually except one case which was referred by the first case I had. The second case from California was referred by the first case, but aside from this they came in not because of the congenital dislocation but because of the pain.

Dr. Porter's question is a very pertinent one. I made a statement which I should not have made. My intention was to put on a spica bandage just for the purpose Dr. Porter mentioned. We do not let the orderly or internes take our patients back to bed. The traction is maintained and the bed is brought to the operating room; the traction is applied at once. I, myself, and the assistant see that the traction is correct and we take the patient to the room.

As to the type of operation, a number of years ago the best result from a congenital dislocation I ever saw was in one of Dr. Davis' cases, in which he made a new acetabulum and put the head in. It was a beautiful result, but my own experience in digging out bone has not been a happy one and therefore I do not believe it is necessary or desirable to take out bone and make a new acetabulum. You can hold the head better by turning down a flap. You do not have a raw surface which endangers motion. I think the results show that you do not get the limitation of motion that you get in cases in which you dig out a new acetabulum. I do not believe you should put the head back in the old acetabulum because I think if you get the head back you get a stiff hip. I have seen no atrophy of the head. I have some radiograms taken after two years which I will be glad to show.

THE CAUSES OF AND CHANGES IN LOOSE BODIES ARISING FROM THE ARTICULAR SURFACE OF THE JOINT.*

BY D. B. PHEMISTER, M.D., CHICAGO.

It is generally accepted that osteocartilaginous bodies in joints arise from three sources—from the articular surfaces of the ends of the bones, from the synovial lining of the joint, and from the diseased tissue which develops in arthritis deformans. It is sometimes difficult, however, to determine from which of these three sources the loose body has arisen, particularly when the examination is made years after the date of detachment and when subsequent changes in the joint have occurred, partly reparative at the site of origin, obscuring it, and partly hyperplastic, from the mechanical irritation of the body in the joint. The loose bodies also change gradually after liberation in the joint and all tend to approach a common morphology. The fibrocartilaginous portions proliferate, increasing the size of the body, and in general make it more spherical. The necrotic elements, consisting of articular cartilage or bone, may gradually be absorbed and replaced by fibrocartilage, which in turn may calcify or ossify, so that its original composition may be completely altered and its shape and size greatly changed.

It is the purpose of this paper to deal mainly with the causes of and changes in the bodies arising from the articular surfaces, touching on those from the two other sources only as they are related to the first.

Loose bodies arising from the articular surface of the ends of the bones constitute the most frequent and important group. In the great majority of cases they occur singly, and when multiple rarely number more than two or three. The knee is most often affected, being the seat of the lesion in at least 90 per cent. of cases. The body comes from the lateral surface of the mesial condyle in the great majority of instances. The patella is occasionally the source, but bodies arising from the external condyle or from the tibia are extremely rare. The elbow is the only other joint in which this type of loose body is at all frequently found, the detachment being almost always from the capitellum. In a few instances the hip and shoulder have been affected. The smaller joints are almost never

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involved. The condition is limited largely to males. In 196 cases reviewed in the literature there were 182 males and 14 females. The onset is usually during either late adolescence or the first decade of adult life and is extremely rare past middle life. The earliest case that I have seen was in a girl at the age of twelve. The affected persons are generally in good health at the onset of the process and give no history of previous joint disease. There is rarely a history of infection in other parts of the body which might be a causative factor in the joint condition. A single joint is practically always involved, except in the knees, where the condition is bilateral in a small percentage of cases. The bodies arise from the articular surface and consist either of articular cartilage alone, or, what is commoner, of articular cartilage with a piece of underlying bone attached. They are usually oval and biconvex in outline and vary considerably in size according to the point of origin. The surface of detachment, when composed partly of bone, usually possesses a greater degree of convexity than the articular surface, which remains practically unchanged.

There has been voluminous discussion, extending over the past half century, concerning the cause of this type of loose body and the processes by which separation is brought about. Since Monro ([1]) first noted, in 1726, that a loose body in the knee had a corresponding defect in the condyle of the femur, traumatism has been considered the important causative factor by the majority of observers. This view is greatly strengthened by the frequent history of trauma. In a considerable percentage of cases there is, however, no history of injury, or it has been so slight that it apparently could not have resulted in fracture and detachment of the loose body in a normal joint. This has led to extensive search for other causes for the detachment. Broca ([2]), in 1854, expressed the view that repeated traumatism, too slight to produce fracture, resulted in disturbed nutrition, with subsequent necrosis and loosening of the affected area. This view was strengthened by the presence of arthritis deformans in some cases. Paget ([3]) thought that there was first a non-suppurative or "quiet" necrosis, with subsequent sequestration and exfoliation of the area.

An active interest in the causes of the condition was aroused by the publication of Koenig ([4]) in 1888. He reported cases with pathologic examinations and called attention to the frequent absence of a history of trauma. His conclusion was that, while some of the loose bodies from the articular surfaces are broken off by trauma, the majority are detached by a disease process of unknown nature, which he designated osteochon-

dritis dissecans. The name implies that the process is inflammatory, but bacteriologic examinations were not made, and his gross and microscopic findings failed to show active inflammatory changes either in the synovial linings or in the loose bodies. He states definitely that the noxæ are unknown. In fact, in a review of his cases, very grave doubt arises as to whether two of them belong to this disease. In each the hip was the joint affected and the head was detached from the neck near the region of the epiphyseal line. Separation in this region as a result of osteochondritis dissecans has not been reported by others, and the pathologic findings were much like those which are commonly found at operation for non-union following fracture of the neck or epiphyseal separation.

The theories that have been advanced to explain the condition have been formulated almost exclusively from studies of bodies from two sources, namely, from the lateral articular surface of the mesial condyle of the femur, bordering on the anterior portion of the intercondylar groove, and from the capitellum of the humerus. Instead of a chronological review of the literature, I will take up the most important theories and discuss the points in favor of and against each.

1. Intra-Articular Fracture. The usual contention is that, as a result of impaction of opposing articular surfaces, particularly when combined with flexion or rotation, a piece is chipped off one side and either goes free into the joint or remains partly attached, only to be freed subsequently by a minor violence. In other cases, the pull of ligaments detaches bone at their points of insertion. Barth ([5]) was the particular champion of the traumatic theory. He studied a large number of specimens and concluded that they originated entirely in this way. He thought that the pull of ligaments on their points of insertion was a more frequent cause than chipping off from the impingement of articular surfaces. A point in favor of the view that ligamentous pull may produce a loose body is the fact that the posterior crucial ligament is inserted to that portion of the lateral surface of the mesial condyle which becomes detached and forms the loose body; but the fact that cartilage alone, to which the ligament is not attached, may come away is against the theory.

There are a number of points which favor the theory of detachment by impingement of opposite articular surfaces. In many instances there is a history of trauma with impaction, either alone or combined with rotation or hinge motion in the joint. Kappis ([6]) has pointed out that the knee and the elbow, which are the common seats of classical loose bodies, are the two joints in which such combinations of motions may occur. In this way the tangential and rotating forces may act on the

convex surface of the condyle and fissure or partially or completely detach portions of the articular end. He thinks that loose bodies arising from the patella are the result of tangential forces. To explain the traumatic origin in the absence of serious injury, he assumes a predisposition as the result of disease or congenital disturbance of the articular cartilage. This would explain the bilateral occurrence in the knees in a certain proportion of cases. Articular cartilage has no nerve supply and the underlying spongy bone is extremely insensitive. This should permit of the occurrence of fracture with little or no accompanying pain and without consciousness of serious injury. Too little attention has been paid to this fact. With the joint opened under local anesthesia for the removal of loose bodies, I have chiseled off marginal osteophytes and small chips of cartilage and bone from the articular surface, apparently without producing any pain.

The occasional finding of fissures in loose bodies, which run for variable distances and in some instances almost completely separate them into two fragments, favors the traumatic theory.

It has long been held that the tibial spine plays a role in the production of loose bodies from the lateral surface of the mesial condyle. Freiberg ([7]) has shown that in certain cases the mesial tubercle of the tibial spine is abnormally prominent or is elongated by the presence of an osteophyte. Roesner ([8]) considered the lesion an impression fracture resulting from impingement of the intercondyloid eminence on the lateral surface of the mesial condyle. He thought this came about just before complete extension of the knee, at which time there is a slight involuntary rotary movement (Schlusskreiselung). If this exceeds the physiologic limits, the mesial condyle can come abnormally far backward and lateralward and impinge on the intercondyloid eminence of the tibia. This brings a bluntly pointed object in contact with a convex surface, and the result is either a localized detachment of cartilage from underlying bone or a subchondral impression fracture, leaving the bony chip attached to the cartilage. He studied the mechanism of origin and concluded that impaction of the opposing flat or concave surface could not produce the injury, and that such a shaped piece could come only from injury by a cone shaped object. Evidences of crushed trabeculæ were found in the bony portion of the specimen which he examined. Failure of union of the damaged portion with its bed completes the picture of the disease.

An objection to Roesner's explanation is that in many specimens that have been examined there is absence of evidence of crushing fracture of the trabeculæ; also it would be more difficult to apply the theory to loose bodies from other places, particularly from the patella. In the elbow

joint, displacement of the radius might be assumed, bringing the elevated margin of its concave end in contact with the capitellum in much the same way as the tibial spine is brought in contact with the mesial surface of the lateral condyle. Roesner's explanation deserves careful consideration, although he offers only one pathologic study in support of it.

The points against the traumatic theory are numerous. Aside from the absence of a history of sufficient trauma, the condition is bilateral in a small percentage of cases where the loose body arises from the lateral surface of the mesial condyle. This occurs too often to be the result of coincident injuries and suggests a disease background for the disturbance. In some of these cases there has been a history of injury in one knee, but not in the other. Buchner and Rieger [9] have studied the structure and estimated the strength of the bone and cartilage of the articular ends, and have concluded from mathematical calculations that it is impossible to detach portions of such size and shape either by ligamentous pull or by direct contact of opposing surfaces. Third: the site of detachment in definite intra-articular fractures is usually not the same as that of the classical loose bodies. In Charcot's joint of the knee, chips are not infrequently broken off as a result of excessive trauma consequent to ataxia and to the loss of the protective sense of deep pain. However, they never originate from the lateral surface of the mesial condyle or from the inferior articular surface of the patella, but nearly always from the margins of the tuberosities of the tibia. At the elbow, the capitellum may be the site of origin of either a classical loose body or a frankly traumatic fragment produced by violence. The fragment, however, in the latter condition is apt to be much larger than in the former, as shown by the report of Lindem [10]. A much commoner seat of intra-articular fracture at the elbow is the head of the radius, and classical loose bodies practically never originate from it.

Another strong point against the purely traumatic origin of loose bodies is the behavior of the fragment in case of frankly traumatic intra-articular fracture. Such a chip, with a fresh fracture surface, almost never remains free in the joint, but becomes attached to synovial membrane, usually in a recess of the joint. I have seen several instances of detached fragments of the head of the radius and of the external condyle in the elbow, of tibial spines and of marginal fractures in Charcot's joint in the knee, and the pieces have always become attached to the synovial lining. In experiments on animals performed by Hildebrand [11], Barth, Rimann [12] and others, it has been impossible to produce permanent loose bodies in the joint by detaching fragments of either cartilage or

cartilage and bone. In eight such experiments which I performed on the knee joints of dogs, all of the bodies became attached. They are usually absorbed and disappear completely in a few months. In dogs, pieces of bone and cartilage, chipped off and reinserted in their beds, became reattached in practically every instance.

Histologic studies of removed bodies, whether completely detached or partially adherent to their beds, no matter how early after the onset of symptoms or liberation in the joint, have failed to show the presence of a fresh fracture surface over the entire extent of separation. There has always been found a fibrous or fibrocartilaginous covering over at least the major portion of the surface, which antedated the liberation of the body in the joint. In some instances there have been giant cells and evidences of absorption, but usually the surface is covered by a layer of fibrocartilage in varying stages of maturation.

2. Trauma Damaging Blood Supply, Leading to Necrosis with Subsequent Gradual Sequestration and Extrusion into the Joint. Axhausen ([13]) was for a long time the particular champion of this view. He assumed that, as a result of impaction from the opposing articular surface, the blood vessels to the part are damaged, either with or without partial fracture, according to the severity of the violence. This leads to necrosis of the area supplied by the damaged vessels. Instead of creeping substitution of the necrotic bone, or bone and overlying cartilage, as would result in case of aseptic bone necrosis in other regions, there forms a zone of absorption resulting in gradual separation and eventual extrusion of the dead portion into the joint. Axhausen thought he found support for this view in his experiments for the production of arthritis deformans, in which he killed articular cartilage or cartilage and underlying bone by chemical and thermal agents. In no instance did he succeed in producing loose bodies. He assumes that in all loose bodies, whether completely or partially detached, the bony portion, when present, is entirely necrotic and the cartilaginous portion partly so. However, he reports few cases, and none of them certain, in support of his views. The incorrectness of this theory as an explanation of all loose bodies is demonstrated by the fact that in quite a number the bone or bone and overlying cartilage has been found almost entirely alive, although bony separation was complete or almost so and had existed for a considerable time before the loose body was removed for examination. The following case shows conclusively that necrosis of the bony portion is not a prerequisite to separation of the loose body.

Case 1. Male, age 21. Always in good health, no history of previous injury or trouble of any kind with the knee. While lying asleep on a bed, face down, with the legs projecting over the edge in the hyperextended position, the patient was suddenly awakened with a severe pain in the left knee, and on palpation a loose body was felt in the lateral portion of

FIG. 1. CASE 1.
Roentgenogram taken two hours after detachment, showing loose body and defect in patella with its margin sclerosed.

the joint. He entered the Presbyterian Hospital immediately. A roentgenogram (Fig. 1) showed a bean-sized shadow at the upper limit of the patella and a corresponding defect in the inferior articular surface of the patella. The margin of the defect in the patella was evener and denser than that to be expected in case of recent fracture, as if cortex had had time

to form since the bony separation. The body was removed at operation two days later. There was bloody fluid in the joint.

Pathologic report: The body (Fig. 2) consists of a layer of articular cartilage and of attached underlying bone, each of about the same thickness. It corresponds in outline to the inferior articular surface of the patella and measures roughly 2.0 cm. vertically by 1.5 cm. transversely by 0.8 cm. in thickness. The cartilage presents a fresh fracture surface about the entire margin by which it was attached to the remaining cartilage of the patella. The bony surface is slightly roughened and is covered by a thin fibrous layer. Nowhere is a fresh fracture surface to be made out on

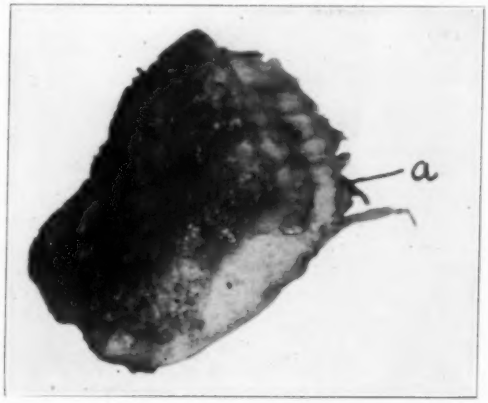

FIG. 2. CASE 1.
Photograph of specimen, showing surface of separation and fresh fracture surface in the cartilage (*a*).

the bony portion. The articular surface of the cartilage appears normal to the naked eye. The gross appearance indicates that the body has been undermined for a considerable length of time and that it was attached only by its cartilaginous portion, which has been freshly fractured. This was probably as a result of the inferior margin of the patella being caught over the superior margin of the articular surface of the femur while the limb was in the hyperextended position.

Microscopic examination of a section through the middle of the body (Fig. 3) shows a bony portion composed of cortex and underlying trabeculæ, that are normal in configuration, with the great majority of the lacunæ filled by living bone cells. At the lower limit of the section

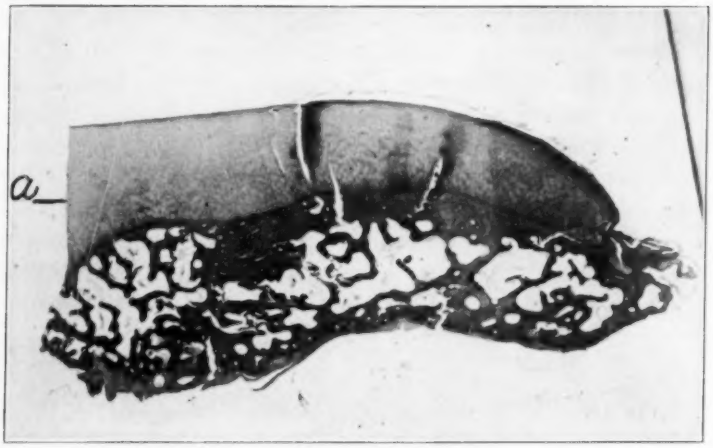

FIG. 3. CASE 1.

Section of body, showing fibrous bony covering and fresh fracture of the articular cartilage (*a*). Tears in the middle portion of articular cartilage are artifacts.

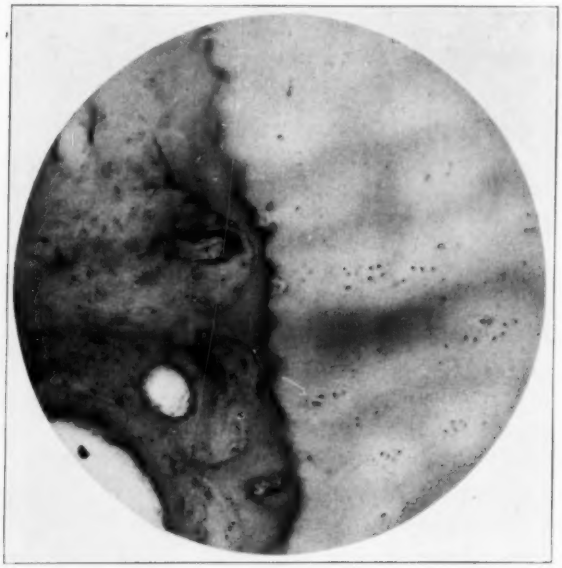

FIG. 4. CASE 1.

Photomicrograph at junction of living bone and articular cartilage.

there is slight marginal necrosis. The rest looks like normal living bone. The cancellous spaces are filled mainly by fatty marrow containing a small number of hemopoietic cells and capillaries that are filled with blood. The overlying articular cartilage is normal in appearance in the greater portion of its extent. Its columns of cells stain normally, and there is no invasion of cartilage from the underlying living bone (Fig. 4). The cells may be reduced in number in places.

FIG. 5. CASE 1.
Surface of separation of bony portion of body, showing fibrocartilaginous covering and underlying living bone.

At its upper limit there is a fresh fracture surface; at its lower limit there is a small marginal osteophyte, but practically no cartilage proliferation with overgrowth upon the bony surface. The surface of separation through the bone is covered in its entire extent by a layer of fibrocartilage, which evidently has been present for some weeks (Fig. 5). There are occasional free shreds of fibrocartilage projecting from the surface, which probably represent broken attachments to the underlying bed. There is slight ossification proceeding from the bony surface into the base of the covering of fibrocartilage, where the cells are arranged in low columns

perpendicular to the surface. No absorptive changes are to be seen along this surface, as if a recent dissecting process had been active, except at the upper limit of the section. Here the fibrocartilaginous covering of the bone is more irregular than over the remaining surface, and there are two small areas of lacunar absorption in the old bone with replacement by new bone.

The first symptoms in this case were produced by the detachment of the body and it was removed from the joint two days later. This is by far the earliest examination of a loose osteo-cartilaginous body that has been made after the onset of symptoms. In the cases where bodies have been found in situ and excised, there have always been symptoms extending over a period of weeks or months. The only evidence of fresh fracture was in the articular cartilage by which the body was adherent to the patella. Judging from the appearance of its fibrocartilaginous covering, the bony portion had been separated for at least some weeks, and no clue as to the method of separation—whether fracture or absorption—was gained from histologic examination of the body. There is no evidence of a crushing fracture, as suggested by Roesner, and if a dissecting osteochondritis led to the separation, its active stage was passed, as there was practically no evidence of lacunar absorption along the surface, and new bone formation, rather than absorption, was found at the junction of bony surface and its fibrocartilaginous covering, except in one small spot. It is somewhat speculative as to just where the blood vascular connection between the body and the rest of the patella was located, but it was probably near the upper margin, where the cartilaginous connection was intact, as there the layer of fibrocartilage appears youngest. All of the bone in the section was alive and was old bone, except along the fracture surface, where a slight amount of new bone had formed. There were no evidences of inflammation in the body, and the blood vessels showed no changes. Most of the larger vessels contained no blood.

Ludloff ([14]) advanced the theory that the loose bodies arising from the lateral surface of the mesial condyle result from injury of the arteria genu media at the point where it perforates the posterior capsule of the joint and before it enters the bone at the seat of insertion of the posterior crucial ligament. The resulting circulatory disturbance leads to necrosis of the area of bone supplied by the injured vessel. The necrotic bone is then gradually separated as the result of insufficient nutrition. There are many objections to this theory. There is no definite anatomic proof that the arteria genu media supplies just the region which becomes separated, and furthermore Nussbaum ([15]) has shown that it is not an end artery. Injury

to an artery by pinching of soft parts is not known to occur in other regions. A number of detached bodies in this vicinity have been found to show little evidence of necrosis in either bone or cartilage. A still further objection is that a similar explanation could not be offered for the detachment of loose bodies in other locations.

Radium Experiments. In order to test the validity of the theory that necrosis of an area of cartilage or of bone and overlying cartilage bordering on the joint is followed by gradual separation of the dead from the living portion, I have killed portions of the ends of the bone in dogs by the insertion of radium into the joint and have observed the changes that followed. Twelve and five-tenths mg. of radium were inserted into the knee joint in the region of the lateral portion of the mesial condyle and left from eight to twenty-four hours in a series of seven experiments. Necrosis of an area of cartilage and underlying bone developed both in the condyle of the femur and in the articular surface of the tibia near its spine. The joints were examined from two weeks to nine months after the insertion of the radium. In no instance was there sequestration of the area of cartilage and underlying bone. The articular cartilage was usually smooth and practically normal in color, although in the older experiments it was somewhat whiter than normal. In a three and one-half months' experiment (Fig. 6), the mesial condyle in the exposed region showed an oval shaped necrotic portion about 1.0 cm. long and 0.75 cm. broad, bordering on the articular surface, and on section it extended in the underlying bone to a depth of 0.5 cm. in its deepest portion. A roentgenogram of a section through the exposed area showed no change. There was no sign of sequestration of the necrotic portion (Fig. 7). Microscopically the necrotic bone in its central portion was entirely devoid of cells and the marrow spaces were filled partly by necrotic débris and partly by a mixture of fibrous and fatty marrow that had grown in to replace the necrotic portion. About the periphery of the necrotic bone there was a definite reparative reaction. Fibrous marrow filled the cancellous spaces, and there was some new bone formation about the surfaces of the trabeculæ, while creeping substitution of the necrotic trabeculæ had commenced by ingrowing new bone without the presence of lacunar absorption by giant cell osteoclasts.

The cartilage overlying the dead bone was necrotic in its entire extent, and toward the middle showed some evidence of irregular surface erosion. At the junction of necrotic and living cartilage, there was very slight evidence of proliferation and formation of fibrous cartilage, which was replacing the necrotic portion.

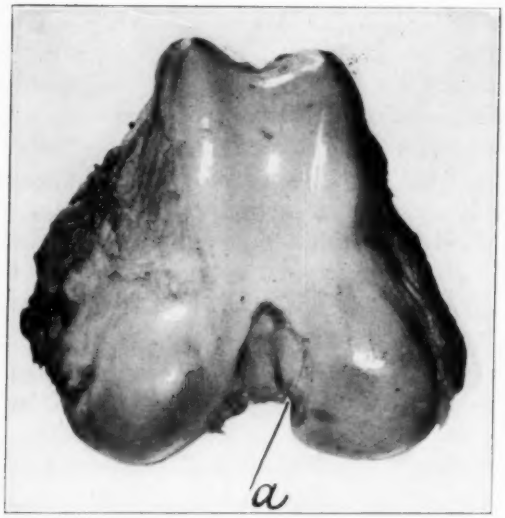

FIG. 6.
Condylar surface three and one-half months after application of radium to lateral margin of mesial condyle (*a*).

FIG. 7.
Roentgenogram of a sagittal section through exposed area of condyle in Fig. 6. No sign of sequestration.

From examination of this specimen, it is evident that, instead of sloughing, there was absolutely no break in the bony connection between living and necrotic portions, and that reparative changes occurred in the living tissue surrounding the dead area, with invasion and a start at gradual replacement of the dead portion by new bone. The changes in the necrotic cartilage were not sufficiently advanced to determine whether it would slough into the joint, become absorbed, or be replaced gradually by fibrocartilage.

3. Embolism. (a) *Fat Embolism.* Buchner and Rieger, after rejecting the purely traumatic theory, assume that loose bodies arise as a result of fat embolism in the vessels supplying the affected region. They offer no evidence in support of their assumption. It would be exceedingly strange if fat embolism were to pick out exclusively the regions of the bone which give rise to loose bodies and produce no other manifestations.

(b) *Bacterial Embolism.* Axhausen ([16]) recently examined histologically a specimen in which there was separation of an intra-articular portion of the distal end of the second metatarsal bone, a condition known in Germany as Koehler's disease, but first described by Freiberg ([17]) in 1914. He expressed the view that it is the result of low-grade bacterial embolism of the artery supplying the area of bone and cartilage which becomes detached. He reported that the old bone and cartilage in the involved area showed evidences of necrosis and that excessive new bone had formed in the process of replacement of the necrotic portions, which increased the density of the region and caused it to cast a heavier shadow than normal in the roentgenogram. From these findings he theorizes as to the cause of loose joint bodies in general, and expresses the view that they also are the result of bacterial embolism. He believes that clumps of extremely avirulent tubercle bacilli lodge in the end arteries supplying the regions from which the bodies arise, resulting in necrosis with very little inflammatory reaction and subsequent sequestration of the damaged portions. He compares the process with the cone shaped sequestration in tuberculous joints, which Koenig explained on the basis of tuberculous embolism. He did not mention the fact, however, that the regions of the knee from which spring the sequestra in tuberculosis and the loose bodies in so-called osteochondritis dissecans are never the same. The tuberculous sequestra practically always arise from the prominent portions of condyles or tuberosities and almost never from the lateral surface of the mesial condyle or from the inferior patellar surface. Also, in only two of the published cases (Freiberg's) of lesion of the metatarsal bone

has there been complete detachment of the body with displacement from its bed. I have examined microscopically the detached bone in a case of this condition.

Case 2. An eighteen-year-old boy, six weeks before admission, leaped from a horizontal bar to a mattress, landing on the toes. This brought on pain and swelling in the affected joint, which had continued and had caused him to limp. The roentgenogram (Fig. 8) showed separation of the cortex of the greater part of the articular surface with some increase in its density and irregular absorption along the line of detachment. The distal half of the shaft was thickened from a layer of periosteal new bone. At operation the separated portion of the articular end of the metatarsal was found attached to the synovial membrane in part of its circumference, and in it there was evidence of considerable bone proliferation. The microscopic section, which unfortunately was cut obliquely to the articular surface, showed proliferation of the bone and of the cartilage about its margins, producing callus and fibrocartilage. There was a certain amount of lacunar absorption going on in the bony portion, but no necrotic old trabeculæ could be made out. There was a small amount of thickening of synovial membrane, with the presence of fibroblasts and round cell infiltration, but there was no leukocytic infiltration nor granuloma formation in any portion of the section. The microscopic appearance was more like that of callus formation from fracture than like inflammation. The periosteal thickening along the shaft of the distal half is more suggestive of inflammation than of fracture, as it is excessive for intra-articular fracture, but the histologic findings are more in favor of fracture than of inflammation. There is more proliferative change in this body, in both its bony and cartilaginous portions, than is ordinarily seen in a classical loose body from the articular surface of a large joint; also, in the latter condition an associated periosteal thickening has never been observed. Nussbaum has recently shown that epiphyseal arteries are not end arteries and has remarked that areas involved from embolism are cone shaped and differ in shape from loose bodies.

4. Constitutional Disturbances Involving Bone. Fromme [18] has expressed the view that loose bodies are detached by the development of a zone of transformation (Umbauzone) such as Looser [19] described in the nutritional disturbances, late rickets and osteomalacia, at points in the bones where stress is greatest. In this condition there is gradual absorption of discs of bone, usually at right angles to the long axis of the shaft, and replacement by a partially degenerated fibrocartilaginous callus.

FIG. 8. CASE 2.

Detached and sclerosed articular surface with adjacent bony absorption and periosteal thickening of second metatarsal, six weeks after injury.

The roentgenogram resembles that of a complete or incomplete ununited fracture. From histologic examination Looser and Seeliger ([20]) claim that the process may start independently of a fracture, either partial or complete. Similar absorptive changes with replacement by fibrocartilage

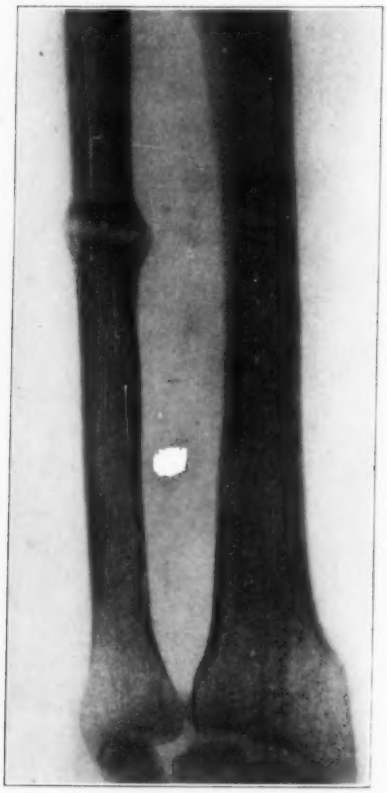

FIG. 9.
Cleft in ulna in fragilitas ossium in adult, the result of lacunar absorption. Fromme claims that it is similar to line of separation in loose bodies.

may occur in fractures in such conditions as osteopsathyrosis, as was first pointed out roentgenologically by Kienboek ([21]). I have seen examples of it also in a case of fragilitas ossium beginning in adult life (Fig. 9)

and in localized osteosclerosis fragilis in a ten-year old girl (Fig. 10). In these cases the transformed zones were discovered incidentally by roentgenologic examination. There had been pains in the extremities, but no acute symptoms suggestive of fracture, although it should be remembered that porotic bones fracture from slight violence and sometimes with almost no pain.

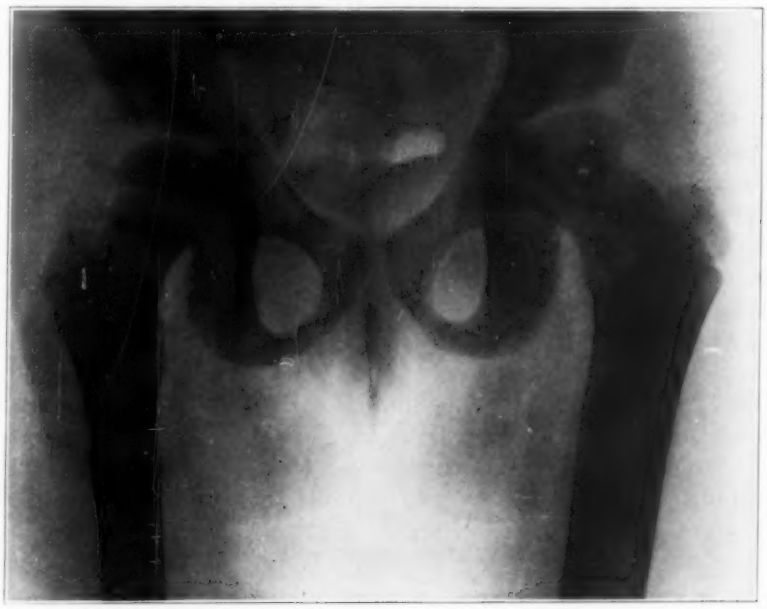

FIG. 10.

Looser's zone of transformation in (*a*) case of localized giant growth and osteosclerosis fragilis, traversing mesial side only of neck of femur. (Patient of Dr. W. L. Brown.)

Fromme has assumed that loose joint bodies develop in individuals who have constitutional disturbance of bone. As a result of partial fracture or of continued stress on the affected areas, zones of transformation develop, leading to bony separation, and subsequent traumatism breaks the fibrocartilaginous connection and sets the bodies free in the joint. He cites the occurrence of bodies bilaterally in the knee in support of the theory. The course of development of zones of transformation has been

followed in the shafts of the long bones by means of the roentgen ray and the process seen to progress from cortex on one side across the entire shaft. No such evolution of loose bodies in joints has as yet been observed. I have examined a zone of transformation excised by Dr. Ryerson from the middle of the femur for correction of deformity in a patient suffering with osteomalacia. It traversed two-thirds of the diameter of the shaft of the bone and was of the shape of a narrow wedge with its base on the convex side of the curvature. Grossly the cleft was filled with a bluish white material. Microscopically this was found to consist of fibrocartilage which was completely broken down in its central portion (Fig. 11). There was osteoid tissue and active new bone formation along either surface, showing that it was in process of repair. There was very little evidence of absorption of old bone to be seen, and there was little evidence of absorption and none of necrosis in the old bone bordering on the margins of the cleft. My opinion is that incomplete fracture is responsible for the formation of these zones. There is always marginal necrosis along fracture lines, with some absorption of the necrotic bone. In conditions of deficient calcium deposition, absorption of the dead bone will occur more rapidly than the formation of completely ossified new bone. This will lead to the replacement of the zone of necrotic bone by osteoid tissue and fibrous callus. Necrosis will result in the central portion of fibrocartilage, as is the case in ununited fracture. The histologic appearance of the covering of the free surface of the body is somewhat similar to that of the material which fills out the space in the zone of transformation, except that there is absence of necrosis in its superficial portion. Looser's zones of transformation have been observed only in the shafts of the bones in definite instances of the diseases in which they occur, and no case of associated loose body in the joints has been recorded.

Fromme claims that the condition known in surgical literature as os acetabuli is also due to the formation of a zone of transformation at the uppermost portion of the rim of the acetabulum as a result of slight traumatism from impaction of the head of the femur. This is different from the anatomical os acetabuli, which is a small prismatic center of ossification in the acetabular margin, located at the junction of its iliac and pubic portions. He found that the surgical, or, as he calls it, roentgenologic os acetabuli, occurred with much greater frequency in patients suffering with softened bones, as rickets, coxa vara, and coxa valga. In no definitely recorded instance has an os acetabuli become completely detached and free in the joint. This condition is observed at the age when loose bodies most commonly develop. In the following case the coxa valga possibly

FIG. 11.

Microscopic appearance of a zone of transformation or cleft in osteomalacic femur.

led to the separation of the upper margin of the acetabulum, but the bony fragment retained its soft parts attachment.

Case 3. Male, age 25, had worked for a number of years at a bottling machine, using the right leg constantly in a semi-flexed position. For three years he has had pain in the right hip and has limped a portion of that time. No history of trauma, other than that associated with occupation. A roentgenogram (Fig. 12) shows a bean-sized shadow (tear drop) at the upper limit of the acetabular margin, separated by a ragged zone from the adjacent bone. A moderate coxa valga is present and it is probable that the impaction of the head against the acetabulum lateral to the normal location has resulted in marginal fracture.

5. Local Osteo-Arthritis. Loose bodies from the articular surface and arthritis deformans are sufficiently often associated to suggest the existence of a disease process responsible for both of them. The first symptoms usually point to the loose body, and generally there are no deforming changes by the time it becomes freed in the joint. If it is removed comparatively early, the joint nearly always returns to normal. If, however, the loose body remains for years, the constant irritation sets up an arthritis which may be markedly deforming and which should be regarded as a secondary change. Rarely loose bodies may be removed and deforming arthritis follow. There may even be secondary loose bodies of the same nature as the first. As yet no microscopic pathologic evidence has been presented which justifies the acceptance of the view that the zone of separation between the loose body and its bed is produced by an absorptive or a dissecting process of an inflammatory nature. The lacunar absorption which may be seen along the surface of the bony portion in specimens that are examined early might well be considered as secondary to fracture, since it is not an infrequent finding along fracture lines in case of delayed or non-union. Axhausen's [22] case showing active absorption at the junction of cartilage and bone did not present evidence of inflammation, and previous partial separation from traumatism cannot be excluded. Microscopic evidence of "osteochondritis dissecans" in the act of producing separation of the bony portion has never been reported. I have seen multiple loose bodies in the elbow with deforming arthritis, where the roentgenogram showed a defect in the capitellum, and one of the bodies removed at operation consisted of bone and cartilage and corresponded roughly to the defect, while the other bodies resembled those found in arthritis deformans. They probably were enlarged fragments ground off from either the loose body or the margins of its defect in the capitellum.

FIG. 12. CASE 3.
Os acetabuli (*a*) in case of coxa valga.

Axhausen ([23]) believes that Legg-Perthes' disease is the result of bacterial embolism of a vessel of the capital epiphysis of the femur, with subsequent necrosis of bone and articular cartilage, but with little or no bacterial proliferation and inflammatory reaction. He thinks the same process in other joints produces loose bodies, but does not mention their occurrence in Legg-Perthes' disease, either in its early or late stage. Figure 13 shows the roentgenogram of the left hip of a patient thirty-seven years

FIG. 13.
Old Legg-Perthes' disease with two large loose bodies in hip joint.

old, who at the age of six had the beginning of symptoms like those of Legg-Perthes' disease. A limp followed, but the hip was painless until five years before examination, since which time the limp has been worse and the joint painful. In the roentgenogram, the head is flattened and the neck shortened and broadened and there are evidences of marked

deforming arthritis, with two shadows suggesting large loose bodies in the joint. These were removed, and at operation marked marginal lipping was found on acetabulum and head. Examination of the loose bodies showed them to be composed of irregular layers of fibrocartilage, which was very extensively calcified. The early lesion was followed, as is common in these cases, by arthritis deformans, and the loose bodies had apparently arisen late in the disease from detachment of overgrown portions of cartilage.

Changes in the Bodies after Separation. After detachment from its bed, the loose body may remain in situ for an indefinite period, held either at its margins by unbroken portions of cartilage, or, as at the knee, by the posterior crucial ligament, or (what is less important) at its base by fibrous bands which form across the zone of separation and connect it with the underlying bed. Fisher ([24]) and Axhausen ([13]) reported cases in which the detached body became permanently reattached and was in process of transformation at the time of the pathologic examination. This occurrence is probably more frequent than statistics would indicate. It is conceivable that a certain percentage of cases cure themselves in this way.

With the loose body in situ, the fibrocartilaginous coverings of the two surfaces of separation, being more or less intimately connected, may increase in thickness, so that it is lifted out of its bed and projects beyond the level of the articular surface. In other cases, bodies coming from the lateral portion of the mesial condyle of the femur are attached only by fibers of the posterior crucial ligament. Many years may pass, three to five not infrequently, before these connecting bands are broken. As long as they are present, the body usually retains a blood vascular connection and marked transformative changes may occur in it while in situ. The original bony portion, in such bodies as have been excised before complete detachment, has been found necrotic in some cases and alive in others. Colvin ([25]), Sommer ([26]), Kappis ([6]), and others reported cases in which the old bone was alive, and I have seen four such instances. With time there is gradual transformation in the bony portion, when blood vascular connection is adequate. The old bone may be gradually absorbed and replaced by new bone, whether the old bone has or has not undergone necrosis from previous nutritional disturbances. The arrangement of trabeculæ in the transformed bony portions is quite different from that in the original bone.

The articular cartilage in the attached bodies may present no change,

but in the older cases there are usually varying degrees of necrosis and transformation. Disappearance of nuclei in the columns of cartilage is common, and invasion with absorption by vascular buds of fibrocartilage from the underlying bone is sometimes seen. Fissures may form in necrotic portions, probably as a result of minor violence. I have seen the cartilage much more necrotic than the bone, where the body has been attached by a pedicle for a long time.

The following case illustrates the changes which may occur in a body composed of both cartilage and bone, which remained partially attached for three and one-half years.

Case 4. B. W., male, age 28. Three and one-half years before examination, patient stepped in a hole, twisting the right knee, and fell to the ground. He experienced considerable pain in the knee and limped for several days. He was then free from symptoms for six months, when the knee again became painful. Since then there have been occasional attacks of sharp pain in the joint, followed usually by soreness and occasionally by swelling which lasted for a few days. Examination showed a healthy young man with slight limitation of motion in right knee. Roentgenogram (Fig. 14) showed a detached piece of the lateral portion of the mesial condyle of the femur, measuring 2.5 cm. transversely and antero-posteriorly, with a narrow, even zone of separation. The density of the shadow of the spongy bone and cortex in the loose body is slightly less than that of the remaining condyle, due to the absence of overlapping. In the side view there is an irregular break in the bony shadow in its interior half. Operation May 19, 1922. The body was movable, but was found partially attached about its margins to cartilage, to posterior crucial ligament, and to the underlying bed. When it was excised, there was some bleeding from the bed.

Pathologic examination: The body is biconvex disc shaped. Its antero-mesial end is detached except for part of its cartilaginous connection and is movable on the larger piece. The cartilaginous surface is smooth except in two places, one near the middle and the other at the junction with the small attached nodule, where it is fissured. The bony surface is nodular and possesses a thin, motley soft-parts covering. A short stub of posterior crucial ligament is attached to one point of the lateral surface of the body. Microscopic section cut through the main body and the small attached portion (Fig. 15) shows the two portions to consist of cartilaginous and bony layers. The small body at the antero-mesial end is composed of a bony center, which is entirely surrounded by a layer of

FIG. 14. CASE 4.
Loose body partially attached to mesial condyle three and one-half years after injury and onset of symptoms.

FIG. 15. CASE 4.

Section of main body with attached bony fragment (*a*). Cartilage fissured, surface of separation overgrown by fibrocartilage and new trabeculæ of bone in cancellous spaces.

cartilage. This is connected by an irregular necrotic bridge with the articular cartilage layer of the large body. The articular cartilage of the large body shows evidence of considerable necrosis with disappearance of its cells, most marked in its peripheral portion. The fracture surface of the bony portion of the loose body shows extensive changes. It consists of a combination of old and new bone and shows definite evidences of bony transformation. There is much new bone formation, especially along the surface of separation and to a considerable extent in the interior regions. The cancellous spaces are filled mainly with a fibrous marrow, richly supplied with blood vessels. The cells of the old bony portion are necrotic or necrobiotic in places, but in others are definitely alive. Bony cortex with attached overlying articular cartilage is practically normal in configuration in some regions, while in others it has been mainly absorbed and replaced by new bone. There are areas in which all of the old cancellous bone has been absorbed and has been or is being replaced by new bone. Islands of cartilage with enchondral ossification are numerous, although there is considerable direct new bone formation by fibroblasts (Fig. 16). The covering of fibrocartilage along the bony surface shows marked degenerative changes in the peripheral portion and slight evidences

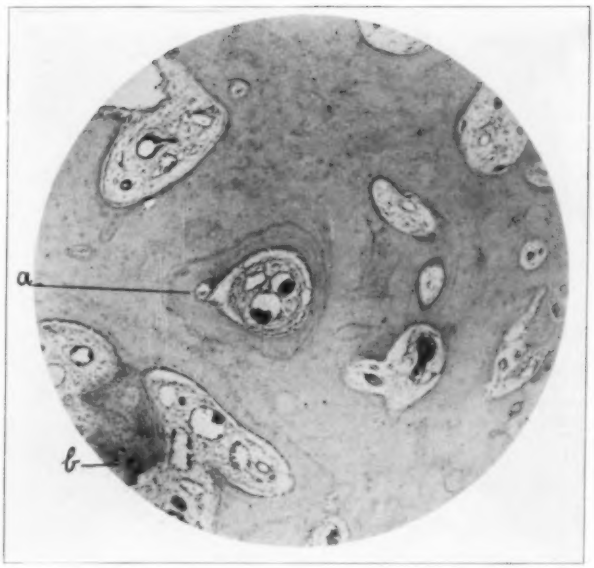

FIG. 16. CASE 4.

Old bone, partly degenerated. New bone forming by both fibrous (*a*) and enchondral (*b*) ossification.

of ossification in its deeper portion (Fig. 17). The fibrocartilaginous covering appears to have arisen along the bony surface from fibrous tissue of the cancellous spaces and is not an overgrowth from the articular cartilage about its margins, since there is little evidence of proliferation of articular cartilage about its edges. A section through the insertion of the fragment of posterior crucial ligament shows a number of small arteries and veins, indicating that it was the main source of blood supply of the body.

FIG. 17. CASE 4.
Bony surface of separation with fibrocartilaginous covering (*a*), degenerated along its surface.

In this case the traumatism to the joint was considerable and was a definite etiologic factor. The histologic examination showed ample blood supply to the body, which entered by way of the pedicle. Articular cartilage was more necrotic than bony portion, but it showed very little transformation and substitution by newly formed fibrocartilage. On the other hand, new bone formation in the bony portion was marked and much of the old bone had been replaced by new, although most of the remaining old bone had not undergone necrosis. This is a further example

of complete bony separation of a body without total necrosis of its original bony portion. There were no signs of inflammation present.

Changes in the Bodies after Liberation in Joint. After complete liberation of a body in the joint, all of the bony portion undergoes necrosis, but the cells of the fibrocartilaginous covering of the surface of separation receive sufficient nutrition from the synovial fluid and largely remain alive. They gradually proliferate and the body slowly increases in size. The condition is analogous to tissue culture in vitro. According to Carrel ([27]), connective tissues in culture do not obtain from blood serum the materials necessary for the synthesis of protoplasm, but demand for that purpose certain substances contained in embryonic tissue and secreted by leukocytes and possibly by other cells. It would probably be found true that synovial fluid in joints is lacking in these materials. The loose body irritates the joint and usually sets up a proliferative change in its synovial membrane. The source of the growth stimulating substances or trephones for the loose body might be the proliferating cells of the synovia or leukocytes poured out as a part of the mild traumatic inflammatory reaction. After years the fracture surface may be found covered by a layer of fibro-cartilage which is as thick as that of the articular cartilage. The articular cartilage slowly undergoes further necrosis and surviving portions show little tendency to proliferate. It is gradually absorbed and replaced by fibrocartilage, and in old specimens there may be difficulty in determining which is the fracture surface and which the articular surface. The necrotic bony portion may also be gradually absorbed and replaced by fibrocartilage (Fig. 18). Bands of fibrocartilage form, which during the period of active growth may run irregularly. Calcification occurs regularly in the deeper portions of the fibrocartilage and to some extent in the original articular cartilage. It may occur in layers. An extremely pathologic type of new bone may be laid down, which is lamellated but contains no bone marrow. The surfaces of the old bodies are usually nodular and the cells of the fibrocartilage radiate toward the periphery. Along the surface there may be a thin fibrous covering with the cells running parallel to the surface. It may be extremely difficult to distinguish them from osteocartilaginous bodies arising from the synovial lining, as histologically they may resemble each other closely. The bed from which arose the body from the articular surface may be so filled out that it is unrecognizable either on roentgenologic examination or at operation.

The following case is an example of the marked changes which may be seen after many years of complete detachment.

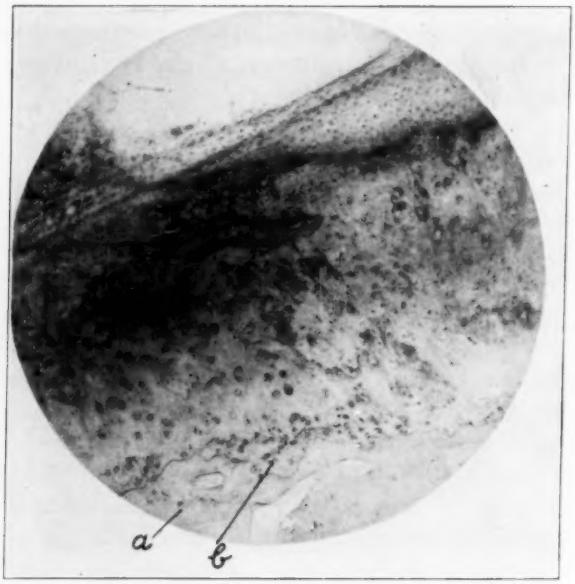

FIG. 18.

Loose body of six years' standing, composed of necrotic bone and of proliferating fibrocartilage. Bone (*a*) is being absorbed and replaced by fibrocartilage (*b*).

Case 5. H. L., male, age 42, had a loose body in left knee joint for 14 years. There was no history of trauma. Patient complained of repeated attacks of pain in the joint and had felt a loose body since soon after the onset. He stated that it had about doubled in size in the past twelve years and was usually palpable in the suprapatellar region. Upon opening the joint, the body was found free and was removed. Pathologic report: The body was biconvex, roughly almond shaped, and pointed at one end. Its dimensions were 3 cm. × 1.75 cm. × 1 cm. It presented a slightly curved, smooth articular surface and a roughened surface of separation (Fig. 19). There had been marked overgrowth along the surface of separation, with the formation of a broad, irregular ridge about the margin of the articular

FIG. 19. CASE 5.

Photograph shows old articular surface (*a*) surrounded by ridge of tissue (*b*) that has formed since detachment of the body.

surface. Cross section at the middle of the body Fig. (20) showed it to be very hard throughout. There is a shallow groove at either side of the bluish-gray portion corresponding to articular cartilage, which has maintained fairly well its original shape. The marked overgrowth of cartilage along the surface of separation is irregularly laminated in its peripheral portion, where it is light yellow in color. Microscopic examina-

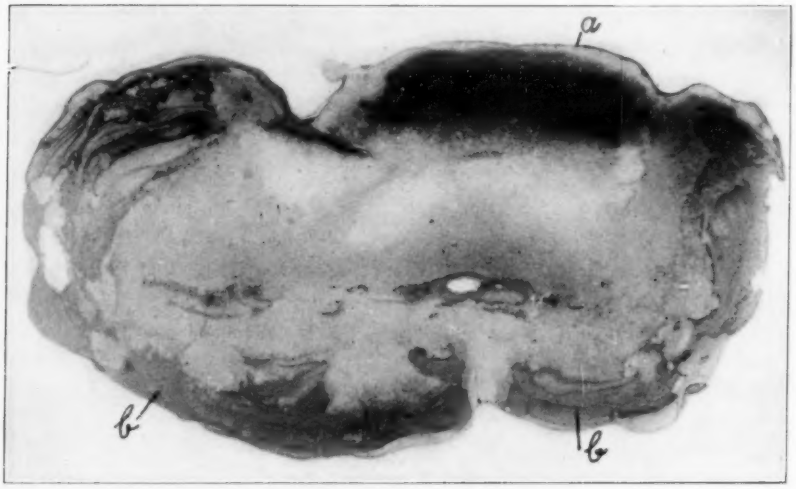

FIG. 20. CASE 5.

Cross section of body, showing region of old articular cartilage (*a*) and hypertrophied portion with irregular cortex of newly formed bone (*b*).

tion of the cross section showed the region corresponding to articular cartilage to be composed of irregular columns of cartilage cells, which are arranged at right angles to the surface. The deeper portion is calcified, while the superficial portion consists of hyaline cartilage. There is a very thin surface layer of fibrocartilage. Nuclei are everywhere more abundant than in normal articular cartilage. There has evidently been almost a complete transformation of the area. The central portion of the section consists of degenerated and calcified cartilage, which is extremely poor in cells. There is no bone to be seen in this region. The peripheral

portion of the remainder of the body, which corresponds to the surface of separation, consists of an irregular layer of proliferating tissue composed of fibrocartilage, calcified cartilage, fibrous tissue, and bone (Fig. 21). The arrangement is extremely irregular. Along the greater part of the surface there is a layer of richly cellular fibrous tissue, but at intervals

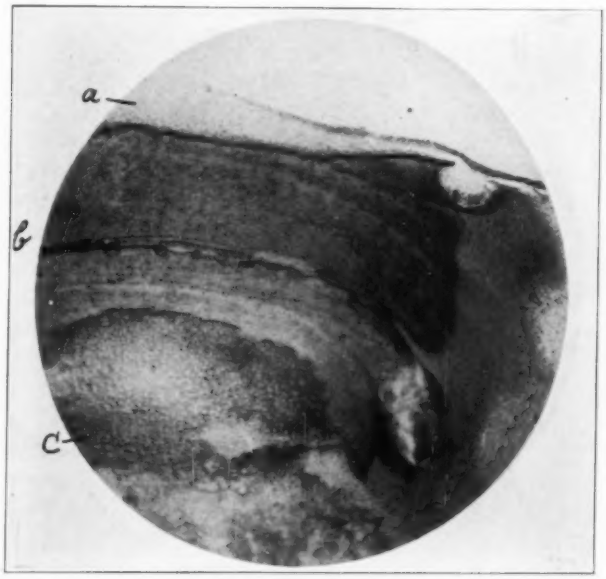

FIG. 21. CASE 5.

Photomicrograph of cortex shown in Fig. 20 with outer fibrous layer (*a*), lamellæ of atypical bone (*b*), and partly calcified fibrocartilage (*c*).

there are nodular projections composed of proliferating hyaline fibrocartilage. Beneath the surface layer there is an irregularly interrupted cortex of extremely pathologic bone. It consists of irregularly interrupted laminæ rich in lacunæ, most of which are occupied by bone cells. This pathologic bone seems to be forming mainly along the surface by direct ossification of the fibrous covering. Interiorly it is in contact with calcified cartilage and shows slight evidences of growth. Ground section

and special canalicular stains show canaliculi connecting the lacunæ in some places. The original body in this case was probably composed only of articular cartilage, although there may have been some attached bone which was gradually absorbed and replaced by fibrocartilage. There are no old bony trabeculæ along the line of junction of the old articular cartilage and of the later formation of calcified cartilage, but there are

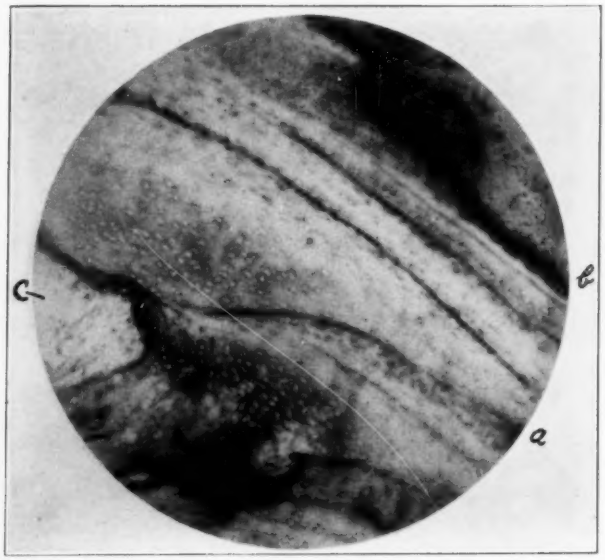

FIG. 22. CASE 5.

Lamellæ of atypical bone (*a*) forming partly by fibrous ossification (*b*) and partly through cartilage (*c*).

in this region a few lamellæ of avascular bone which formed subsequent to detachment. Were it not for the well-preserved outline of the original articular cartilage with the proliferation located about its margins and along the surface of separation, it would be very difficult to distinguish this body from an osteocartilaginous one arising from a tag of synovial membrane.

SUMMARY OF ETIOLOGY. From a consideration of the evidence for and against the various theories of causation of the classical loose bodies that have here been enumerated, we are forced to the conclusion that an entirely satisfactory explanation has not yet been offered. The lesion does not seem to be a complete intra-articular fracture. If it were, fragments detached from sources other than the lateral surface of the mesial condyle of the femur, the patellar surface, and the capitellum of the humerus should as regularly persist as loose bodies in the joint, which is not the case. Incomplete or subchondral fracture passing between cartilage and bone or through bone is a more plausible explanation, although there are many objections to it. Non-union would have to follow and the cartilaginous bridge would be severed subsequently, either by trauma or by absorption. Nutritional disturbance in the partially detached portion would favor the occurrence of non-union, but the partial fixation would greatly increase the chances of union with its bed. It is possible that the cause of non-union is general and is not understood, as is the case in a certain percentage of ununited fractures of the shaft. Articular cartilage is devoid of nerve supply and there is extremely little sensation in the cancellous bone which it overlies. It is possible that a fracture of the articular surface might result from undue violence and be accompanied by little or no pain. Such injuries would easily be overlooked or forgotten, and this would account for the frequent absence of a history of trauma of such violence as would ordinarily be considered necessary to produce such changes.

Primary necrosis of the portion which becomes detached has been shown not to occur in at least a certain percentage of cases, as in Case 1 of this series, and, as demonstrated here, portions killed experimentally by radium do not slough into the joint.

Arterial embolism, either fatty or bacterial, cannot be accepted as the cause, for there is no plausible reason why it should be limited to the particular regions of the ends of the bones from which the loose bodies spring. There has been no definite pathologic evidence presented in support of embolism. Constitutional disturbance of the bones may play a role, but not enough clinical or pathologic data are at hand to warrant one in classifying the zones of separation as identical with the clefts of Looser which form in the shafts in such diseases as late rickets and osteomalacia.

No inflammatory changes have been observed in loose body, and "osteochondritis dissecans" has never been observed during the stage of separation of a fragment consisting of bone and cartilage.

SUMMARY OF PATHOLOGY. After the body becomes loosened, degenerative and regenerative changes go on in it, both while it is attached by a pedicle and after it is completely liberated in the joint. While attached by a pedicle, it may be nourished through blood vessels and the degenerative changes may be slight. However, both articular cartilage and bone tend to gradual necrosis. Proliferative changes may be considerable during this period. They consist mainly in the formation of fibrocartilage along the surface of separation, but where bone is present there may be also considerable new bone formation, most marked in the cancellous spaces along the zone of separation, and the old bone which becomes necrotic may gradually be replaced by new bone. After complete liberation in the joint, all bone which has had a blood vascular circulation becomes necrotic and there is still further necrosis and calcification in the articular cartilage. However, the fibrocartilage and fibrous tissue along the surface of separation receive sufficient nutrition from the synovial fluid and proliferate, causing a slow increase in size of the body. The fibrocartilage gradually absorbs and replaces the necrotic articular cartilage and less rapidly the necrotic bone, so that years after separation the original constituents of the body may have completely disappeared. Calcification takes place in the fibrocartilage and an extremely pathologic type of new bone may be formed in layers in its superficial portions.

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THE AGE FACTOR IN HYPERTROPHIC ARTHRITIS.

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THE subject of arthritis has been most efficiently confused by several and contradictory nomenclatures under which different investigators have described its several forms. For example, so-called degenerative arthritis is also described as hypertrophic, and proliferative arthritis is alternatively designated atrophic. This contradiction is due to the fact that one group of writers bases terminology upon the changes occurring in the soft parts, the other group upon bone changes. Thus the atrophic type of joint disease is characterized by proliferation of the soft structures and atrophy of the bone. Other names have also been applied to the same conditions. Various types of joint lesions of infectious origin have been likewise confused.

The more recent studies have attempted to reduce this confusion and have combined different varieties into major types. Thus we find that Swaim (1), basing his claims on the work of Goldthwait, Painter, and Osgood, classifies arthritis as Infectious, Atrophic, and Hypertrophic. The infectious type he describes as presenting no bone change. Little information of importance has been added to that presented by Rosenow in his original studies of this type (2). It is recalled that Rosenow found the infecting organisms present in larger numbers in the periarticular structures than in the joints,—that is, in the relatively avascular areas where the capillary supply really ends. He doubted the power of the organisms to grow in the free circulation. This localization must be considered a possible source of continued irritation in the production of capsular lipping.

The atrophic type of Swaim's classification presents proliferation of soft structures, widening of the joint, destruction of cartilage and bone, and resulting ankylosis and deformity. He finds this variety predominantly in the visceroptotic type of individual, the slender neurotic, whereas the hypertrophic variety occurs in the heavy, sluggish, hypopituitary patient. The latter type is also said to occur as a rule in old age or premature old age. It is associated with trauma which decides the

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location of the lesion for which the diathesis is responsible, and is often monarticular. This type Swaim finds rarely in the slender type of back. It is painless in onset and is often well advanced without symptoms. It frequently causes joint pain through mechanical irritation.

In summarizing, the conclusion is drawn that the infectious type handles the bone salts well, the atrophic type either does not get bone salts or is unable to handle them, while the hypertrophic type has overabundance of capacity to handle them. Treatment is advised in accordance with these precepts.

Ely (3) combines the atrophic form as described above with all infectious types, dividing the entire category of joint inflammations into two great types which he designates as types *one* and *two*. In type one he includes all frank infections and the atrophic type. The pathologic changes in general consist of rarefaction of bone and thinning of cartilage. There is no new bone formation.

Of type *two*, the hypertrophic type, "Chronic Rheumatism of the Elderly," Ely states that the primary pathology is necrosis of bone and marrow in the vicinity of a joint. As a result of this necrosis there is a deposition of new bone at the capsular attachments about the joints. Ely states that this hypertrophy cannot be the result of internal bone changes, new bone being laid down about the necrotic area. The cause, he states, is unknown, but in his opinion it may be related to the presence of ameba histolytica about the teeth and gums.

It is of interest to note that in these and other classifications of arthritis the hypertrophic type of lesion is considered as a distinct entity. In each, its relation to advanced age is recognized but considered of secondary importance, and in each classification a different primary factor is invoked.

The present discussion of hypertrophic arthritis is based upon definite bony changes consisting of new bone formation as found in a series of 625 spinal columns examined in the Hamann Museum. The study is limited to the changes appearing in the lumbar portion of the spines because the data were obtained during an investigation made primarily for skeletal anomalies of the lumbo-sacral region; because during the investigation it was observed that this part of the column was especially subject to these changes; and because it is in reference to this region that the X-ray diagnosis of hypertrophic arthritis is so frequently made.

As no method of measuring the exact amount of bone change in the various skeletons could be devised it was determined by visual estimation. One appreciates that this is inexact, but for want of a better method

it has been used. As the estimation upon all the material was made by the same individual during a short period of time, and with no preconceived ideas as to the significance of the condition, the results may be considered as relatively fairly accurate. The degree of hypertrophic change has been charted as minus, one, two, three, and four plus. Typical examples of these grades are shown in Table I.

TABLE NO. 1.

Age		0–19	20	25	30	35	40	45	50	55	60	65	70	75	80	85
S		9	10	24	11	43	22	13	3	1	1					
A	—	6	16	18	21	32	8	5		1						
H			6	3	2	2	4									
S				1		5	15	12	16	8	5	3	1			
A	+			2	4	22	29	24	16	7	5	2	2			
H				2	2	7	5	5	4	1			1			
S						0	1	1	7		3		3			
A	+					1	2	6	18	10	9	8	7	3		1
H	+					3	8	7	5	6	6	4			1	
S	+															
A	+							1			1	2	1			
H	+						3	3	6	3	5	2	3		2	1
S	+															
A	+															
H	+									1	1	1	1	1	1	

TABLE NO. 1. Degree of hypertrophy (lipping) in different types of skeleton, by age periods. The table gives the actual number of skeletons of slender, average, and heavy type (S. A. H.) in each five-year period. The degree of hypertrophy (lipping) is indicated as minus, one, two, three, and four plus.

The accuracy of the age data may be accepted as reliable. The ages as obtained from the records of the City Hospital and Morgue have been checked by the Department of Anatomy with the estimated age of the body and with the age changes determined by members of this staff upon cranial sutures, long bones, symphysis pubis, etc. When there is a discrepancy between the different estimates such fact is noted.

On account of the work of Goldthwait, Painter, Osgood, Swaim, and others in relation to arthritis, and their classification of subjects into slender and heavy anatomic types, I attempted during the examination of these skeletons to divide them into similar groups. I found however that there were many "in-between" individuals and that it was necessary

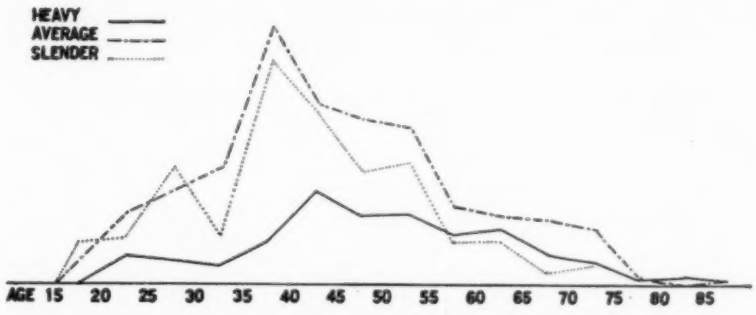

CHART NO. 1, showing the relative number of skeletons of the slender, average, and heavy types as found in the material examined. The skeletons are divided according to age into five-year periods. Actual numbers are not inserted because it is purely the relative number which one desires to emphasize. Dotted line represents the slender type, the interrupted line the average, and the continuous line the heavy type.

to add to these types an "average" type. Chart 1 shows the proportion of these three types as found among the skeletons examined. The material is also divided into five-year periods according to age. It is seen that the average type predominates, the slender running a close second, while the heavy type is noticeably less common than either of the others. In addition it is noted that the slender type disappears from the chart fifteen years earlier than do the others. This is probably due to the fact that in advanced age the erosion of the vertebral bodies and the lateral lipping increases the width of the body in relation to its height, the segment therefore assuming a heavier type of appearance.

A number of these vertebræ, particularly those which showed marked hypertrophic changes, presented an appearance resembling vertical compression of their bodies. This was found particularly in the lowest dorsal and lower lumbar segments and suggested that the throwing out of the lipping processes might be evidence of an attempt to bolster up a weakening structure. In practically all of the columns examined, hypertrophic changes were more marked in the lower lumbar segments and progressively less so from there upward. In other words, the segments which are under the greatest mechanical stress present the greatest amount of lipping. Exaggeration of the lumbar curve increases the strain and increases the lipping.

The grading into anatomical types of these skeletons was done by visual estimation. This, however, seemed to be a condition which could be checked by measurement. From each group an equal number of specimens was chosen at random. These were measured accurately to

determine the relation of width to height of the vertebral bodies. This gave the so-called vertebral index of each lumbar segment, the formula being $\frac{\text{height} \times 100.}{\text{width}}$ The column index was obtained by averaging the individual indices in each. The columns of each type were then averaged. The one departure from the usual procedure of measurement was that in obtaining the height of a segment the greatest height was taken rather than the measurement at a certain fixed point. This was done in order to obtain a measurement that would correspond most closely to that obtained in a radiogram. As a result it was found that by actual measurement those subjects classified by observation as of the slender type possessed a vertebral index of 69.46, the average type of 63.06, and the heavies of 59.82. It would seem, therefore, that the vertebral index furnishes an exact method of separating skeletons into what I consider as the anatomic types as designated by the above-mentioned writers. Until we are supplied with a method more exact than that of observation it may be acceptable.

Admitting then the deficiencies in exactness of the methods used, but maintaining that, as these were used impartially in all the subjects examined, the results are relatively reliable, the investigation has developed a much more definite relation of hypertrophic changes to age than has been appreciated. Table 2 presents this relation. It is seen that up to

TABLE NO. 2.

AGE		0–19	20	25	30	35	40	45	50	55	60	65	70	75	80	85
S	—	100	100	96	100	89.5	57.6	50	11.6	11.5	11.3					
A	—	100	100	90	84	58.5	20.3	13.7		5.6						
H	—		100	60	50	16.5	20									
S	+			4		10.5	39.6	46.5	61.6	88.5	55.3	100.	25			
A	+			10	16	40	74.3	66.7	47	38.6	33.5	16.6	20			
H	+			40	50	58.5	25	33.5	26.5	9.25			20			
S	±					1.5	2.6	3.5	26.6		33.3		75			
A	±					25	5.3	16.7	53	55.6	60	66.6	70	100		100
H	±						40	46.5	33.5	54.25	50	57.3			25	
S	+															
A	+							2.7			6.5	16.6	10			
H	+						15	20	40	27.25	41.25	28.3	60		50	100
S	+															
A	+															
H	+									9.25	8.5	14.3	20	100	25	

TABLE NO. 2. Degree of hypertrophy (lipping) in different types of skeleton, by age periods. The arrangement is the same as in Table 1, but instead of actual numbers, the percentage is computed. In the vertical direction the squares represent degrees of hypertrophy (lipping): in the horizontal direction they indicate age periods. In each square then there is inserted the percentage of each type of skeleton for one specified degree of hypertrophy.

the age of 35 there is practically no lipping found. In the years from 25 to 29 inclusive there were five spines showing the first degree of hypertrophy, one of the slender type, two of the average, and two heavy. In four of these five the age estimate based on changes in the other bones was of the next later decade. In the following period there are six individuals showing hypertrophy, four of whom are of the average type. These six also showed general bone changes of an age greater than the chronological age. There was either a mistake in the age as given in the records or there was premature ageing as shown by the general skeletal changes.

During the period between 35 and 40 years there appears a very definite degree of change, and this change is greatest in the heavy type of spine and least marked in the slender. At this age but 10% of the slender type show hypertrophy, while 41% of the average type and 83% of the heavy type present definite change. Note also that 25% of the heavy type have already acquired the second degree of hypertrophy. From this age onward the process increases steadily in all three types of spine. At 45 the heavy type disappears altogether from the minus column. Five years later the average type drops out except for one straggler at 55, and in the following half decade the slender type practically drops out. These facts support the claims of the above-mentioned writers that the heavy type of individual is more prone to hypertrophic changes than is the slender type, but do not support their contention that these changes occur only in advanced age and are rarely found in the slender type. We find the changes appearing a decade later in the slender type but occurring just as invariably. The lipping does not progress to so great a degree, however, as none of this type reaches the third degree of change and the class is poorly represented in the second degree. The average type is poorly represented in the third degree and only the heavy type reaches the fourth or extreme degree of hypertrophy.

In charting these statistics as curves (Chart 2) the number of specimens of each of the three types occurring in each five-year age period was taken as one hundred per cent. The percentage of each type in each degree of hypertrophy was multiplied by the degree and the products added. The sum of the products of each type gave the graphic value of that type. Thus in the age period designated as 35, which includes those skeletons of 35 to 39 years inclusive, there are 48 of the slender type. Forty-three or 89.6% of these show no lipping and are considered as of minus degree. They are therefore multiplied by zero. Five or 10.4% show the first degree of change and are multiplied by one. Then 89.6×0 plus 10.4×1

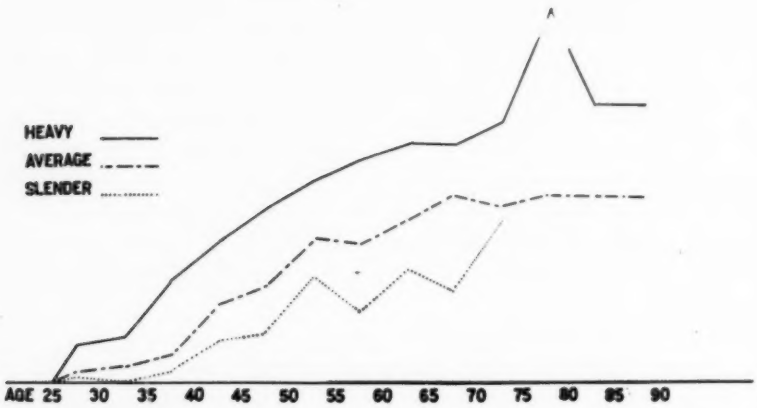

CHART No. 2, showing the relation of hypertrophic changes to age in the slender, average, and heavy types of skeletons. The graphic value of each type at any particular age is obtained by the multiplication of the percentage of the type for that particular age by the degree of hypertrophy (see text). Increase in the number of cases showing higher degrees of hypertrophy causes the curve to mount correspondingly steeply. Dotted line represents the slender type, interrupted line the average, and the continuous line the heavy type.

equals 10.4, which is charted as the amount of hypertrophic change found in those skeletons of the slender type which were 35 to 39 years of age.

Hypertrophic bone changes are much more general and much more closely related to age than has been realized. These changes are practically universal after the forty-fifth year and progress regularly from that time on, no type of individual being immune to the process though there is a distinctly stronger tendency to progress in inverse ratio to the vertebral index of the individual.

A bone change due to age alone should show a steady continuous curve. In addition to the progressive rise in the curves above we notice more or less distinct irregularities. These must be accounted for by a more erratic cause than age. We know that continuous mechanical irritation may produce hypertrophic bone change. We have seen these changes at the lumbo-sacral articulation in cases of spondylolisthesis when the remainder of the spine showed none whatever. We also know that bone and joint structures respond in identical manner to irritation whether it be of traumatic, chemical, or infectious nature. We may, therefore, suppose that while age has a universal and continuous influence, there are other factors which accentuate the progress of the condition. But is not age itself pathologic, and where can the dividing line be drawn?

Is there any difference between the bone hypertrophy of age and that due to other pathologic influences?

We note in Ely's description of Hypertrophic Arthritis (3) that the primary pathology of the condition is necrosis of bone in the vicinity of a joint with a resulting disposition of new bone about the necrotic area and at the capsular attachments.

Todd (4), in that section of his work on age changes of the symphysis pubis referring to skeletal changes in general, speaks of a non-pathologic type of lipping as "the ossification which occurs in tendinous and ligamentous attachments to bone after the age of 35." He describes an osteophytic growth in the sacrotuberous ligament which is definite and constant enough in relation to age to warrant its use as an age indicator. This is not found before 35, but is progressive thereafter. At the age of 50 he notes a rarefaction and erosion of the symphysial face, with erratic ossification which is possibly pathologic.

We have the clinician describing the bone changes of the "Chronic Rheumatism of the Elderly" as necrosis in the vicinity of a joint with irregular laying down of new bone at the capsular attachments, while the anatomist describes age changes in the bone as rarefaction and erosion with erratic ossification. The difference then must be one of degree or of arrangement. Theoretically the age influence is exerted steadily and continuously but is necessarily affected by general habits of life and general physical condition. Its "lipping" should be "regular." The pathologic influences, variable in severity and continuity, exerting an erratic and spasmodic influence, should result in bizarre lipping with sclerosis and osteoporosis of the bone, not seen as a result of age influence alone.

Clinically, in a patient over 35 years of age, how can we know whether a certain amount of lipping as shown by the roentgenogram is the result of age or of other pathologic cause? Since the partial dissemination of that group of conditions formerly known as "rheumatism," we have been pleased to diagnose as hypertrophic arthritis any condition in which the roentgenogram shows a suspicion of lipping at the capsular line of a joint. This explained the pain and was easily demonstrated to the patient. But is such a diagnosis sufficient in light of the fact that we so often find these changes when there are no symptoms present?

Very naturally, as the ligamentous structures about a joint become ossified their function is diminished. Even at 35 one does not ordinarily retain the resilience of 20. These ageing joint structures become more and more susceptible to injury, and more and more slow in recovery from injury, whether the injury be produced by trauma, infection, or chemical

results of metabolism. The influence of age must be reckoned with. The appearance of hypertrophic changes in a radiogram may signify only that the subject has passed the fourth decade of his life.

Clinically, age must be considered as a factor in every case of hypertrophic arthritis, and premature age may be shown in joint tissues as well as elsewhere. Added to this factor there may be others, such as mechanical irritation from faulty posture; developmental or traumatic defects; irritation due to chronic infection localized, as noted by Rosenow, in the relatively avascular areas at capsular attachments; or chemical irritation from toxic absorption of metabolic products.

Some individuals are peculiarly liable to hypertrophic arthritis of the polyarticular variety which spreads over a large number of the more peripheral joints and is crippling and disfiguring. The entire bone end changes as though it had lost the influence which maintains its form. What is the relation of this diathesis to the bone and joint conditions found in Paget's disease, leprosy, and Morven's form of syringomyelia, and in gout; to scirrhus, keloids, calcified scars, nonunion of bone and soft tissues? What is the cause of Perthes' and Köhler's diseases, late rickets and osteomalacia? Why is lipping of bone much less marked in tuberculous patients and why is there such a variability in the amount of bone callus thrown out in different patients? Perhaps for the same reason that there are both atrophic and hypertrophic types of Charcot joints. Given a patient presenting joint symptoms, whose roentgenogram shows hypertrophic bone changes, what may be ventured by way of prognosis and treatment?

The bone changes are permanent. The age influence is progressive. If there be such a factor as a bone forming diathesis this is presumably permanent. Age and diathesis are both active and predisposing elements. The various irritants—mechanical, chemical, infectious—serving to accentuate the process, are aggravating factors. These are subject to proper attack. Since even the advanced stage of hypertrophic change may be present without evident symptoms, it is possible, by removing the exciting factors, that existing symptoms may be relieved. The prognosis then is by no means entirely unfavorable.

Treatment must be directed toward the identification and removal of aggravating etiologic factors, more than one of which may be concerned in the individual patient.

Mechanical irritation, often following postural strains of flat foot, lordosis, or occupational habit; local joint irritation arising from focal infections so commonly found about the mouth, throat, nose, and prostate;

chemical irritation following toxic absorption due to visceroptosis, intestinal stasis, and chronic fatigue; these are the more common of such factors. Search must be thorough and is frequently tedious, but results are often strikingly worth while. Danger lies in the fact that, encouraged by results obtained through the removal in certain cases of one of these factors, this one may be over-estimated in its importance. Survey of recent literature seems to indicate that such a danger does exist.

Summary.

Hypertrophic bone changes represent the reaction of bone and joint structures to the wear and tear of life. They are more pronounced in certain types of individuals and are aggravated by continued local irritation of any character.

These changes rarely occur before the thirty-fifth year of age but are progressive thereafter. They may exist to a marked degree without clinical symptoms. Being the result of metabolic changes rather than the cause, their presence does not prohibit relief from such symptoms.

The bone changes are permanent. Treatment of the symptoms must be directed toward the removal of all possible sources of continued irritation. These include mechanical strains, focal infections, intoxications, and absorptions, any or all of which factors may be present in a given case.

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DISEASES OF THE BLOOD VASCULAR SYSTEM OF THE EXTREMITIES. AN EXPERIMENTAL STUDY.

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THE clinical manifestations of a disordered blood vascular system of the extremities are too numerous to enumerate. It is only necessary to mention varicose ulcers, ischæmia, ischæmic contracture, erythromelalgia, aneurysm, endarteritis obliterans, thrombophlebitis, and the various sorts of spontaneous gangrene to include a considerable proportion of all cases presenting themselves to any surgical clinic. Furthermore, at the present time, little is being done for these conditions other than the institution of measures affording temporary relief or radical operations for removal of the diseased extremities. No attempt will be made to discuss the many clinical manifestations of blood vascular diseases of the extremities. The results of some experimental investigations will be briefly outlined and certain general principles, together with their possible clinical application, will be discussed.

1. *Experiments in which the Effects of Ligation of the Primary Arteries of Extremities Were Studied.*

Dogs were used for these experiments. The anatomy of the arterial tree in the dog is such that complete obstruction of the iliac and hypogastric arteries always leads to manifestations of impaired circulation of the posterior extremity. The magnitude and character of the changes observed varied in different animals due to variations in the adequacy of the collateral circulation.

In some animals the obstruction of the iliac and hypogastric arteries resulted only in an altered physiological condition. The posterior extremity was not completely paralyzed but there was an abnormal stiffness of the muscles. The extremity was used for walking, but after the animal used the extremity for a brief period it became incapable of supporting the body-weight. If the animal was permitted to rest for a period, the extremity could be used, but soon again became useless. This condition

usually gradually improved until the extremity became normal. Examination of the muscles showed no evidence of any anatomic change. There was no degeneration. There was no fibrosis. This condition is believed to be one of ischæmia. It is a condition in which the flow of arterial blood is reduced below that required for the normal muscle metabolism of activity, but in which the flow of arterial blood is not reduced below that necessary to maintain viability of the tissues of the extremity. The disturbance in function is a phenomenon of fatigue and is not a result of anatomic pathologic muscle change.

In other animals, in which the hypogastric and iliac arteries were obstructed and in which the collateral circulation was less, gangrene developed. In some instances only the skin of the distal portion of the extremity became necrotic. In others both the superficial and deep structures became gangrenous. If both the skin and muscles became necrotic, the necrosis of the skin was always more extensive than that of the muscles. Very curiously the individual muscles usually became entirely necrotic or remained totally viable. It thus happened that a totally viable muscle might lie alongside a totally necrotic muscle. In some instances, however, only the distal end of a muscle became necrotic, the proximal end remaining viable. In such an instance the line of demarcation between the dead and living muscle was sharp and exactly corresponded to the line of separation of the paralyzed and non-paralyzed muscle. This is an important point because it shows that there is no such thing as a true ischæmic total paralysis of muscle. Furthermore, muscle tissue either remained viable and retained its normal anatomic characteristics or it became completely necrotic. In no instance was muscle fibrosis observed as an early result of only arterial obstruction. We do not believe that the condition originally described by Volkmann as "ischæmic paralysis" is due to permanent arterial obstruction.

2. *Experiments in which the Effects of Temporary Arterial Obstruction Were Studied.*

It was found that the effects of temporary occlusion of the primary arteries of the extremities were different from those of permanent occlusion of these arteries. If the arteries of the extremity were permanently occluded and necrosis of tissue followed, it was most extensive in the superficial tissues. If the arteries of the extremity were occluded by clamps which were removed after a certain period and necrosis of tissue followed, it was most extensive in the muscles. This shows that skin is more susceptible to a continued diminished blood supply than muscles, and that

muscles will survive for a shorter period than skin a complete absence of blood. The iliac and hypogastric arteries of the dog were occluded by clamps for a period of 12 to 17 hours and then the clamps were removed. The circulation was re-established in all of the larger arteries. The muscles were examined and it was found that there were many small areas of necrotic muscle. (I wish particularly to emphasize this finding because it has an important relation to the results obtained in another experimental study to be discussed later). The muscle which remained viable retained its normal physiologic properties. The necrotic muscle became slowly replaced by fibrous tissue. There was no marked inflammatory reaction and no rapidly developing fibrosis and contracture.

3. *Experiments in which the Effects of Alteration of the Blood Circulation in a Single Muscle Were Studied.*

In these experiments the rectus femoris muscles of dogs were used. The muscle was dissected completely free from all attachments except its origin and insertion and its artery, vein, and nerve. In each experiment a tight ligature was tied about each extremity of the muscle. This procedure resulted in the muscle being isolated from all circulation of blood except that entering through a single artery and returning through a single vein. The muscle also was surrounded on all sides by normal living tissue. It was found that ligation of the artery supplying the muscle usually led to no anatomic or physiologic change. In a few instances the muscle became necrotic and was completely absorbed. The same results followed ligature of both artery and vein except there were more instances in which the muscle became completely necrotic. If the vein alone was obstructed the muscle became swollen, hard, and extensively infiltrated with blood and polymorphonuclear leucocytes. A severe acute inflammatory process rapidly developed and the entire muscle was transformed into a contracting mass of fibrous tissue. This process developed only if there was complete venous obstruction and no arterial obstruction.

4. *Experiments in which the Frequency of Gangrene Following Obstruction of the Primary Arteries Alone and Following the Obstruction of the Primary Artery and Vein of an Extremity Were Studied.*

Rabbits were most suitable for these experiments. It was found that obstruction of the iliac and hypogastric arteries of one side was followed by gangrene of the corresponding posterior extremity in 75% of twenty experiments. In eighteen experiments it was found that obstruction of the iliac and hypogastric arteries and the common iliac vein of one side was

followed by gangrene in 33% of the experimental animals. In two experiments the common iliac vein was injected with barium sulphate paste in such a manner as to produce enough venous obstruction to cause persistent edema. Subsequently ligation of the aorta in these animals resulted in gangrene only on the side of venous obstruction.

These experiments show that the frequency with which gangrene follows the ligation of artery is decreased by a certain amount of simultaneous venous obstruction, and increased by a greater amount of venous obstruction.

5. *Experiments in which the Effects on the Volume Flow of Blood of the Extremity After Ligation of the Primary Artery and the Subsequent Ligation of the Primary Vein Were Studied.*

Dogs were used for these experiments. The animals were anesthetised with paraldehyd and the experiments were conducted for periods of four to six hours. It was found that ligation of one iliac artery caused the temperature of all the tissues of the corresponding extremity to fall. The fall in temperature was greatest in the most distal tissues. If the arterial obstruction was released the temperature of the tissues immediately rose to that of the tissues of the normal extremity. If the artery was obstructed and the temperature of the tissues of the corresponding extremity was permitted to become constant, then obstruction of the iliac vein always resulted in a further fall of the temperature of all tissues of the corresponding extremity.

The results of these experiments show that ligation of the primary artery of an extremity reduces the volume flow of blood through the extremity, and that this reduced volume flow is further reduced by obstruction of the primary vein of the extremity.

6. *Experiments in which the Intravascular Pressure Distal to the Obstruction of an Artery Was Measured and the Effect on this Pressure of Obstruction of the Concomitant Vein Was Studied.*

Dogs were used for these experiments. The animals were anesthetised with paraldehyd and the experiments were conducted for a period of four to six hours.

It was found that ligation of the iliac artery caused a marked fall in the arterial pressure and a complete disappearance of the pulse pressure distal to the ligature. There was also an appreciable fall in the venous pressure of the corresponding iliac vein. If the iliac artery was obstructed and the intravascular pressure in the arteries and veins of the corresponding ex-

tremity was permitted to become constant, then ligation of the corresponding iliac veins caused immediately a marked rise in both the arterial and venous pressure distal to the ligatures. The rise in the venous pressure was greater than the rise in arterial pressure.

These experiments show that the decreased intravascular pressure of an extremity distal to the obstruction of an artery is increased by subsequent obstruction of the primary vein of the extremity. The fact that the venous pressure is increased more than arterial pressure indicated a diminished volume flow of blood through the extremity.

DISCUSSION.

These apparently disconnected experimental facts may be conveniently discussed as manifestations of a disturbance in the balance of circulation of blood through an extremity. The two fundamental factors of blood circulation through an extremity are: (1) the amount of blood flowing through the extremity in a given time, the volume flow, and (2) the pressure under which the blood is confined in the vessels. Assuming the circulatory apparatus of the remainder of the body is normal, the flow through an extremity depends on the cross-section area of the arteries, the capillaries and the veins of the extremity. Decreasing the cross-section area of either of these diminishes the volume flow. The intravascular pressure in the capillaries is dependent on the relation between the cross-section area of the arteries and veins. Diminution of cross-section area of the arterial tree only decreases the intracapillary pressure. Diminution of the cross-section area of only the veins increases the intra-capillary pressure. Normal nutrition of an extremity demands a certain minimum volume flow of blood and this blood must be in the vessels above a certain minimum of pressure. It is possible that the volume flow may be more than sufficient for life and the intravascular tension below the level for adequate nutrition and gangrene will develop. The decreased intracapillary pressure probably is harmful because of the fact that the blood filters through the capillaries of some areas, while in other areas there is a complete collapse of the capillaries and no blood passes through them. This view is supported by the observation that blocking of arteries for a brief period and then releasing the block is followed by focal necrosis. If the volume flow is more than adequate and the intravascular pressure is inadequate, gangrene may be averted by partial venous occlusion which increases intravascular tension, although such a measure actually decreases the volume flow. Increasing the intravascular pressure keeps all capil-

laries open and thus results in a homogeneous distribution of blood, although the total amount is actually reduced. Venous occlusion, however, which increases intravascular pressure in the capillaries may be harmful if this increase in intravascular pressure is obtained only by reducing the volume flow below the minimum necessary for life. Furthermore, intravascular tension in the capillaries may be increased by local total venous occlusion until actual mechanical rupture of the entire capillary system may occur.

The application of these general principles serves to explain certain clinical phenomena and offers a working basis on which certain advances may be made in surgical therapeutics.

1. *Contractures Due to Disturbances in Circulation.*

There are two sorts of contractures following obstruction of the blood vascular circulation. There is the sort which was originally described by Volkmann in which there is a fracture, usually of the distal end of the humerus. A bandage is applied, and in the course of a few hours the forearm and hand become extremely painful. The bandage is removed and the arm becomes tremendously swollen, tender, painful, and a marked contracture rapidly develops. Examination of the muscles after some months have elapsed shows the muscles converted into a veritable fibrous mass. The characteristics of the early stage of this disease are those of inflammation. The condition is the result of a total obstruction of the venous system without obstruction of the arterial system. In this condition the obstruction of the outflow of blood raises the intravascular pressure in the capillaries until there is a complete disruption of these vessels. The reaction in the tissues is that of acute inflammatory disease, and extensive fibrosis of the tissue follows. The fact that there is no obstruction to the arterial supply is proven by the fact that severe inflammatory process exists. Anemia and severe inflammation are incompatible pathologic processes. Actual necrosis of tissue is not a prominent feature of this condition.

Contracture may follow temporary arterial obstruction or permanent obstruction of an artery which is followed by a severe anemia and a rapidly developing collateral circulation. In this condition there results as a consequence of the temporary reduction of arterial flow focal necroses in the tissue. After the removal of the arterial obstruction or the resumption of arterial flow by the dilatation of the collateral arteries the multiple infarcts are replaced by fibrous tissue. In such a condition there is much less manifestation of inflammation and the contracture develops more slowly.

2. *Gangrene from Ligature of Large Arteries.*

The necessity for ligation of an artery which endangers the viability of an extremity not infrequently arises. The obstruction of the artery throws the burden of supplying an adequate amount of arterial blood for the viability of the extremity on the collateral circulation. The collateral anastomoses increase from day to day. There is, therefore, a critical period in which the blood supply may be inadequate and gangrene develops before a collateral circulation develops. Our experiments indicate that the risk of gangrene may be decreased by producing a certain amount of venous occlusion at the time the artery is ligated by ligation of the accompanying vein also. This procedure in all probability is beneficial because of the fact that it increases intravascular tension and thus leads to a more homogeneous distribution of blood. The ligation of the vein actually decreases the volume flow of blood. It is important, therefore, not to produce so much venous obstruction as to decrease the volume flow below that necessary for life of the extremity. The principle of simultaneous ligature of vein and artery first advocated by Sir George Makins is, therefore, applicable in certain instances, and I believe should be used if the arterial ligation is at such a point as to endanger the extremity and there is not already present evidence of venous stasis.

3. *Spontaneous Gangrenes.*

Spontaneous gangrene results from several varieties of arterial disease. From the viewpoint, however, of possibilities of surgical treatment all diseases of the arteries of the extremities can be divided into two groups: (1) Those in which there is disease of the entire arterial tree, and (2) those in which the arterial disease is confined to a relatively small area in a large artery or to only a branch of the primary artery.

The first group of cases is exemplified by the extensive and extreme arteriosclerosis of diabetes, in which all of the larger arteries of the leg may be calcified and thickened until the lumina of all the arteries are reduced to the size of a small thread. This condition is such that the possibility of restorative measures is very remote. The entire arterial tubage is reduced. The disease is progressive and is associated with a general condition the course of which is relentlessly downward. In some cases of endarteritis obliterans the entire arterial tubage is almost completely obliterated and the circulation of the extremity is reduced to what might be called a sort of filtration process through the small arteries and their collateral anastomosing branches. The correct surgical treatment of these conditions will probably continue to be radical amputation at a level far removed from the line of gangrene.

The second group of cases is best exemplified by thrombosis or embolism of the popliteal artery. In this condition the arterial tubage may be quite normal above and below the obstruction. In a considerable proportion of cases of thromboarteritis the process is limited to a small segment of the popliteal artery. In some instances the process known as endarteritis obliterans may affect only the posterior tibial artery. In these conditions there is a definite possibility of relief by surgical treatment. There is an adequate arterial tubage in the affected extremity distal to the arterial obstruction. The volume flow of blood is not adequate, or the intravascular pressure is not sufficiently great for an efficient distribution of the blood. The problem to be solved in such a case is to get blood through or around the obstruction or make it possible to use the blood which is getting around the arterial obstruction more successfully. To accomplish the former the arterial block must be bridged by a vessel transplant or the collateral branches must be widened. In this connection if the operation of perivascular sympathectomy is to accomplish anything it is my belief that the sympathetic nerves must be operated on at such a site that the result is a dilatation of the branches of artery proximal to the obstruction which supply the collateral circulation, rather than a dilatation of the arteries distal to the arterial block. If it could be established that the volume flow of blood through or around the block was adequate for life and the intravascular pressure was too low for proper distribution of the blood, then improvement would probably follow the production of an occlusion of veins sufficient to balance the circulation and maintain a higher intravascular pressure.

The greatest need in the treatment of disabilities of the extremities due to diseases of the vascular system is methods of examination. Methods of determination of volume flow of blood which are in use by the physiologists have been very little used in clinical medicine. Methods of determination of blood pressure are sufficiently accurate only for estimating blood pressures relatively high and only in vessels which have a considerable pulse pressure. All methods are completely useless to determine the pressure in the pulseless artery distal to an arterial obstruction. There is no method for determination of the caliber or extent of obliteration of an artery. At the present time experiments are in progress to determine the possibility of studying the size of the lumen of the arterial tree by intra-arterial injection of sodium iodide and Roentgen ray studies. If this method is successful, as the experiments promise it to be, then it will be possible to determine the site and extent of arterial obstruction, and it may indicate the distribution of the blood flowing through the artery injected.

It would seem that heretofore attention has been too much centered on the vasomotor apparatus. It is possible that more could be learned if the viewpoint was shifted to that in which the blood vessels were considered as tubes. At least it is certainly more within the grasp of the practical surgeon to consider the circulation of the blood in the extremity as fluid circulating through a system of tubes than to think more or less vaguely of the phenomena of the sympathetics. Every specimen amputated for gangrene and examined by me has shown definite mechanical blocking of the lumen of the artery, and I have never as yet seen a clinical condition of ischæmia which I could be reasonably certain was due to arterial spasm.

DISCUSSION OF PAPER OF DR. BROOKS.

Dr. Nathaniel Allison, St. Louis, Mo.: Our Association is indebted to Dr. Brooks for this admirable presentation of his investigations of the circulation of the extremities. This is especially the fact when we consider how little interest in the circulatory mechanism we are apt to take, and how little we really know about this mechanism and its relation to the various diseases of the extremities that we are in the habit of treating. It is not uncommon in our experience to have to treat *erythromelalgia, endarteritis obliterans, intermittent claudication, ischemic paralysis,* and similar disorders that have their origin in circulatory disturbance. We know enough to treat them empirically and that is about all. Our Association is indebted to Dr. Brooks for giving us a clear explanation of the cause of contracture in what we have been calling ischemic paralysis since Volkmann's time. Dr. Brooks has held open a door that was closed to us, so that we have had a glimpse of many things that we should understand and treat rationally. It is rather curious that even in our text-books there is no attempt to show how much the welfare of the surgery of the extremities depends on the efficiency of the circulation of the blood. I, personally, am much beholden to my associate, Dr. Barney Brooks, for his researches on the circulation of the extremities.

Dr. A. M. Shipley, Baltimore, Md.: It might interest you just to hear this: Many years ago when Bernheim in Baltimore was attempting to treat and cure Raynaud's disease by reversing the circulation, there happened to come into the hospital a man on whom he had done an anastomosis in Scarpa's triangle. He had Raynaud's disease of the upper extremity as well and was in such trouble that he came to Bay View seeking relief. He had a definite gangrene of the foot. Of course, we were anxious to know whether Bernheim had reversed the circulation and so an amputation was done. We found that the anterior tibial vessel was empty and was contracted, and, when it was cut across, no blood came through. The perineal artery bled just a little. The posterior tibial artery was empty. One of the veins was bleeding arterial blood in a rather slow, steady stream. That to us was an interesting observation, although it did not prove he had reversed the circulation, and, as you will know, his results in this operation were in the main disappointing.

DR. W. E. GALLIE, Toronto, Ont., Canada: I feel that we have listened to-day to one of the most masterly presentations ever made before this society. Hearing the paper has confirmed in my mind the impression I had already received from seeing Dr. Brooks' preparations and experimental work that the conception of Volkmann's paralysis described to-day is the one which will ultimately be accepted as correct. I shall not attempt to discuss the paper, but I think the excellence of the work and the presentation merit special recognition from the society. I shall, therefore, move a hearty vote of thanks to Dr. Brooks for appearing before the society with this paper.

(NOTE: The motion was seconded and unanimously carried.)

DR. BARNEY BROOKS, St. Louis, Mo. (closing the discussion): Dr. Shipley stated that a case in which the circulation had been reversed subsequently had an amputation, and that during the operation of amputation it was observed that arterial blood flowed from the severed vein. This fact was believed to be proof of the actual reversal of the circulation.

The fact that arterial blood flowed from the severed vein only proved the presence of an arteriovenous fistula. To prove the actual reversal of the circulation it would be necessary to demonstrate that venous blood flowed from the distal stump of the severed artery.

PROGRESSIVE MYOSITIS OSSIFICANS, REPORT OF A CASE AND A REVIEW OF THE LITERATURE.*

BY ANDREW PRITCHARD MACKINNON, M.D., C.M., F.A.C.S.,
WINNIPEG, MANITOBA.

PROGRESSIVE myositis ossificans is sufficiently uncommon to justify a detailed report of the following typical case:

J. S., male. Farmer, age nineteen, came to the Galloway-Gibson Clinic November 29, 1922, complaining of stiffness of the body and limbs.

He was born in Canada of Slavic parents. His father, previously a robust individual, died of influenza in 1918, at the age of fifty. His only brother died of some fever at the age of nine. His mother is living and well. She states that none of his relatives have been similarly affected, nor did any of them have any deformity of toes or fingers. She further states that the patient has never had any other illness, but that he was perfectly normal up to the age of fifteen, except for the deformity of the great toes about to be described, which was noticed at birth.

The first symptoms followed an injury in May, 1918—that is, when the patient was fifteen years old. He was working with his team in the bush, when a large log jammed against his right thigh. The injury was severe enough that he had to be carried into the house, and he spent the next two months in bed suffering from pain in the right lower extremity. By Christmas of the same year this limb had recovered except for some stiffness in the knee, and he was again at work. However, for two or three months previously he had noticed some difficulty in raising the arms above the head, and the ridges on the back, which now show so well, were beginning to form. Nevertheless, he continued to do a man's work about the farm until May, 1921, when a severe illness began. The symptoms came on gradually with pains in the left hip and left knee, which steadily grew worse and involved the other side, until at the end of two weeks he was confined to bed. His mother states that he suffered severe pain and could not move either lower limb or rise to the sitting position, but that he was able to feed himself. After nine weeks in bed he began to move about again, and very soon the pains disappeared, but the stiffness remained. Since the late summer of 1921 his condition has been stationary.

* Read before the Winnipeg Medical Society, September 21, 1923.

At the time of examination the patient walked with difficulty supported by a cane held in the right hand. The whole frame showed a slight general flexion, suggesting the attitude of paralysis agitans. Some movement was present in the left hip, so that he was able to take short steps. Both knees, the right hip, and the lumbar and thoracic spine appeared to be completely fixed.

FIG. 1.
Back view of patient. Note ridges of bone.

Physical examination and X-ray examination showed the symptoms to be due to masses of bone occupying the situations of various muscles. The head did not show anything abnormal, and was not X-rayed. The jaws could be opened to the full normal extent. The neck was negative except for some pencils of bone in the substance of one of the deep cervical muscles in the left side. The appearance of the back suggested the ridges on a relief map (Fig. 1). A rod of bone about one inch in diameter

extended from the mid thoracic region to the sacrum, joining the spinous processes above, but lying behind them below (Fig. 2). Marked atrophy of the erector spinæ group of muscles was apparent. On each side the latissimus dorsi seemed to have been converted into an irregular band of bone, which extended well into the posterior axillary fold, but not to the humerus (Fig. 2). There were narrow pencils of bone in both deltoid muscles. In the left arm a rod of bone about one-half inch wide and three and one-half inches long extended from the middle of the humerus

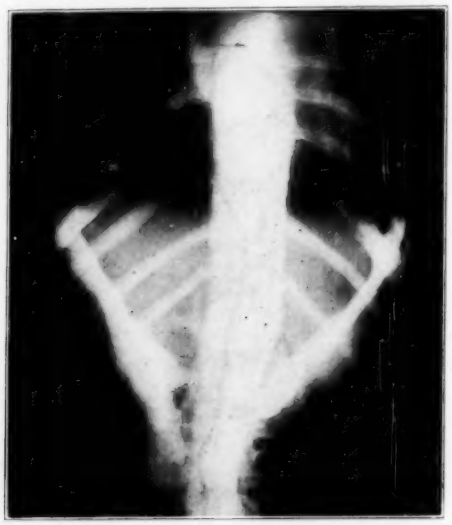

FIG. 2.
A. P. view of trunk, showing median rod of bone, and the bands in situation of the latissimus dorsi muscles.

downward in the direction of the brachialis anticus muscle (Fig. 3). A similar but smaller mass extended upward from the insertion of the triceps. The right arm, both forearms, and both hands were apparently normal.

The lower limbs showed more involvement than the upper. In the right thigh laterally were irregular plaques of bone, but most remarkable was a dense band, an inch and a half wide, extending from the os pubis to the upper part of the femur in the position of the upper margin of the abductor magnus, its density contrasting with the rarefied appearance of the femur. (Fig. 4). The left thigh showed a similar but smaller band in the

FIG. 3.
The Left Arm: Bone formation in brachialis anticus and triceps muscles.

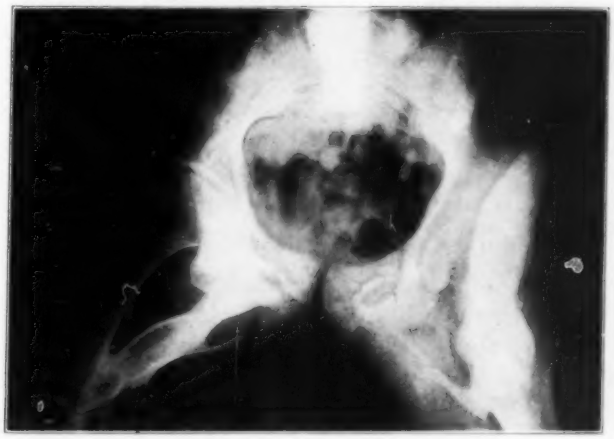

FIG. 4.
The pelvis and upper part of the thigh.

adductor region, and a thick mass corresponding to the upper part of the rectus femoris. Lower down there were other but less dense masses. The upper third of each leg showed some involvement, and there was some new bone formation on the dorsum of the left foot.

Both great toes showed the typical micro-dactylia so often described in this disease. The heads of the metatarsal bones were irregular, and the two phalanges were fused and shortened and in the position of hallux valgus, the tip of this digit being under the middle of the second toe. (Fig. 5).

FIG. 5.

The Feet. Note the irregularity of the head of the first metatarsals, the hallux valgus, and the fusion of the phalanges of the great toe.

The patient was of average intelligence. The genital organs and the secondary sexual characteristics were normal. The blood sugar was found to be quite normal, 0.066 gm. per 100 cc. on one occasion. The basal metabolism was plus twelve, not definitely beyond normal limits. On a meat-free diet no increase in the creatin excretion was observed. The calcium metabolism was not studied. The blood Wassermann was negative. Red and white cells counts and differential counts were not abnormal. During two months' observation the evening temperature was frequently elevated.

Progressive myositis ossificans is rare. In 1918 Rosenstirn (1) of San Francisco collected and summarized one hundred and fifteen cases, the earliest dating back to 1692. Quite recently Nutt (2) of New York has reported a case and summarized thirteen others not included in Rosenstirn's series. In addition, the late Dr. Yasutata Takasaki (3) of the University of Tokyo has reported two cases. With the one here described, therefore, one hundred and thirty-two cases are now on record. A study of the literature seems to justify the following conclusions as to the clinical course of the disease.

The first manifestations are almost always observed during the period of growth, and usually during early childhood. Thus in one hundred and sixteen cases in which the age of onset is stated, the first symptoms were noted during the first year of life in nineteen, and during the first five years in seventy-nine. Twenty-one more began before the age of eleven; eight more before the age of sixteen; seven between sixteen and twenty, and one after twenty. In one hundred and twenty-six cases in which the sex is stated there were seventy-five males and fifty-one females. Heredity does not appear to be important as an etiologic factor. In the great majority of cases the family history is stated to have been negative. There is, however, evidence of hereditary predisposition in three cases. In one (4) the father, who was accidentally killed at the age of thirty-three, had slight changes of myositis ossificans at the time of his death, and had microdactylia of the great toes; in another (5) the father, and in a third (6) the mother, while free from the disease, did have microdactylia. The vast majority have occurred in the white race but one patient was a little negro girl (7)* and two were Japanese (3). Rosenstirn (1) describes a case in a setter dog which, it is of interest to note, had microdactylia. He states that other instances of the disease in the dog have been recorded.

Of special interest from the standpoint of etiology is the frequently associated microdactylia. This curious condition is almost always present. It was first described by Helferich (7) in 1879, and in seventy-one of the one hundred and ten cases since recorded it is stated to have been present, whereas in only five (3.9.10.) is it stated to have been absent. As there is no record of an X-ray examination of the hands and feet in any of these cases the possibility of a slight degree of microdactylia having been overlooked has to be considered. The deformity consists of a shortening of one or more digits, most commonly the great toes, with fusion of

* A case recently described by Barker (Clinical Medicine, 1. W. B. Saunders Co. 1922) occurring in a young negro male, is to say the least, not typical, and is not included in this summary.

the phalanges. The only other congenital anomalies that I have found records of are one case of hypospadias (11) and one of ectopic testes (12).

The usual history is that, with or without traumatism, painful swellings form, which finally become smaller and painless, and are then found to consist of deposits of bone in the substance of muscle. This sequence of events is repeated at longer or shorter intervals, until finally the greater part of the voluntary muscular system is converted into bone, and the patient dies of some intercurrent affection. When the disease begins in babyhood, however, these painful swellings, especially about the head, often subside without the formation of bony tumors, so that the true nature of them is not disclosed until other parts of the body become involved. The rapidity of the progress of the disease varies in different individuals. Thus in three of the cases beginning during the first year of life, the ages of complete incapacity were nine, sixteen, and forty years respectively. Another case began at the age of eighteen, and the patient was completely bedridden seven years later. In contrast with these is the skeleton of a man described by Opie (13), who died at the age of seventy, and had comparatively slight changes, not enough to prevent him from living an active life.

The most striking involvement is in the muscles of the back and proximal parts of the limbs. In at least twenty-five of the recorded cases there was some bone formation in one or more of the muscles of mastication, leading in some cases to great difficulty in feeding the patient. The muscles of the eye, tongue, diaphragm, and the nonstriated muscles escape.

In three of the recorded cases fractures occurred. All healed promptly without excessive callus. Two others had osteomyelitis, running a not unusual course. One was insane, another epileptic, and three showed some evidence of infantilism (1).

The new bone has the histologic characteristics of bone in normal situations. Cartilage has frequently been found associated with the bone. Much work has been done on the pathogenesis of this condition, but no very definite conclusions have been reached. The frequent if not almost invariable association with microdactylia makes it certain that the underlying cause is operative very early in fœtal life. The painful swellings above alluded to are believed by Rosenstirn to be due to hemorrhages into the tissues and he believes that the effused blood with its calcium content starts the first deposit of lime, which in these abnormal individuals indirectly stimulates further bone formation. This view would bring progressive myositis ossificans into harmony with the much commoner circumscribed traumatic form, but it leaves much to be explained. Painter

(14) suggests that there is a diathesis or dyscrasia underlying both forms, differing only in degree. Rosenstirn still further develops his argument by suggesting that just as the microdactylia is evidence of imperfect development of the extremities of the limbs, so the tendency to hemorrhages may very well be due to imperfect development of the extremities of the vascular system, *i.e.*, of the capillaries.

Treatment is unsatisfactory. Exposure to X-rays has been tried. The improvement reported would appear to be no more marked than one would expect from the remissions observed so often in untreated cases. Removal of bone with the hope of mobilizing a joint has invariably proved disappointing, as the bone reforms in a few weeks, and there is no improvement in function. The various glandular extracts (15) have been given a thorough trial without benefit.

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COMPOUND FRACTURES OF THE FEMUR AND OPEN FRACTURES INTO JOINTS.

BY WILLIAM O'NEILL SHERMAN, M.D., PITTSBURGH, PA.

PREVIOUS to Lister's discovery of antisepsis, a compound fracture of the femur or compound joint injury usually resulted either in amputation of the extremity or death, either from sepsis or gangrene. The mortality in the American Civil War from such injuries was very great, and in most cases amputation was done. The mortality and the number of amputations was considerably reduced in the Russo-Japanese and Boer Wars. This was chiefly due to the application of the principles of asepsis. The first few years of the World War showed little or no material improvement, in so far as infection was concerned, over conditions preceding the discovery of antisepsis. This was due to military conditions, the lack of personnel and equipment to render proper operative treatment, the failure of the so-called antiseptics, and the hesitancy to recognize the great value of early and thorough débridement. Wounds and fractures in the Russo-Japanese and Boer Wars were due in large part to the use of high-velocity missiles, while in the World War most of the wounds were caused by high-explosive shells and machine gun bullets, there being a great difference in the gross characteristics of the wounds. While no absolute and definite rules can be laid down, and each case should be considered separately, there are, nevertheless, certain fundamental and basic principles that are applicable in the treatment of all compound fractures and wounds of joints. One must constantly bear in mind the actual etiologic factors producing the injury and the environment in which it occurs, as they influence to a certain extent the method of treatment. Where the trauma consists of a small wound, a local or superficial débridement is usually all that is necessary, while in those in which there is a large wound of the soft parts, an early and thorough débridement of all the devitalized tissues at the earliest opportunity is indicated. In civil practice one does not often find it necessary to follow the radical débridement practised by many operators at the Front. In the rolling, crushing injuries seen in civilian practice, it is most difficult and often impossible to determine how extensive and what tissues should be débrided. One frequently finds extensive areas of bruising and ecchymosis with

thrombosis resulting: it is imperative that such areas be treated conservatively. Past experience teaches one the extent of the débridement necessary of this tissue.

TREATMENT OF TRAUMA OF THE SOFT PARTS.

Excision or débridement of the skin and soft parts can be followed by a primary suture if the nature of the injury is such as to permit of this procedure: this should be done only in selected cases. It is a most vicious and dangerous procedure to attempt this in the vast majority of cases. It should be remembered that the etiology of fractures and joint injuries in civilian life and those of war are often dissimilar, and there is often a different pathological picture present. Primary closure in compound fractures and joint injuries has frequently necessitated amputations and caused death from sepsis. Mechanical sterilization (débridement), followed by chemical sterilization with secondary closure or cicatrization by granulation, is the method of choice in the majority of cases and gives the best end results. Primary suture in femur fractures and joint injuries should be practised only by those of great experience and judgment, and where the patient can be under the close observation of the operator. It is not to be advised or practised as a routine procedure. In selected cases and where carried out by a very skilled surgeon, a primary suture of joint wounds has given excellent results. Experience and judgment are often the determining factors upon which to base proper procedure. Satisfactory alignment can usually be secured by traction and proper splinting, so that internal fixation is only to be used where expectant treatment has failed.

TREATMENT OF FRACTURE.

Should there be extensive comminution, internal fixation with plates, screws, etc., is not advisable and is usually contra-indicated, unless the comminuted particles can be removed and 50% of the circumference of the fractured ends brought into close approximation. The loose and detached fragments should be removed. The wound following débridement should be left wide open and no attempt to close either the skin or soft parts should be made. The Carrel method, using 0.5 sodium hypochlorite, should be instituted at once and the wound allowed to close by granulation or secondary suture. A partial closing of the wound interferes with the proper carrying out of the Carrel method and is frequently a factor in the

failure to control infection. Should there be a long spiral or oblique fracture present, transfixation with one or more machine tap screws should be done, no plates being used. Should the fracture be transverse and in poor alignment, or in malposition, it should be reduced and fixed with a vanadium steel plate and tap screws. Constricting and encircling bands are not satisfactory. In all compound fractures, if foreign bodies such as screws and plates are used, no attempt whatsoever should be made to close the wound. The plates and screws in all cases should be removed in from four to five weeks and the wound allowed to close by granulation. We are quite aware that this method of treatment is contrary to the general practice and belief of most surgeons, but, nevertheless, we have followed this technique in a large number of cases since 1916 with most excellent results and freedom from infection. In the early part of the War steel plates and screws were used with most disastrous results, necessitating orders forbidding such practice. One naturally asks why the early results were so disappointing. This can readily be answered—it was due to the failure to properly débride the wounds or blind faith in and failure of the so-called antiseptics, such as iodine, mercury salts, the carbolic group, and the failure or inability to provide proper and suitable conditions for the treatment of such cases. In the latter part of the War, most of these conditions were satisfactorily corrected, except in times of great military activity. The infections were not due to the plates and screws, *per se*, but were caused by the failure to perform early and thorough débridement. This caused the traumatized tissues to slough and infection followed. Another contributing factor was the general failure of the antiseptic to inhibit or assist in combatting infection. At the time the plate is removed, a slight separation of the superficial cortex may be seen. This should be removed by curettage. It is understood that if satisfactory alignment of the fracture can be secured and held in a reasonable position without internal fixation, then one can direct attention toward the treatment of the wound. If the proper débridement is done and the Carrel technique is properly carried out, there is little or no danger of infection. We have no hesitancy in making this statement. This method of treatment has been the means of saving many extremities from amputation and possibly of averting death. We feel that it is a great scientific step in advance and not difficult to carry out. The same general principles of wound treatment are applicable in joint wounds. Following débridement, primary closure can be done if, in the judgment of the operator, it is safe to do so. A thorough exploration for foreign bodies should be made and if they are found, removed. Loose and completely detached bone fragments should

be removed. Should the fragments be displaced but viable, they can be reduced or, if necessary, fixed in normal position with nail or screw. Non-hand-contact technique is desirable. Active mobilization should be started within three or four days and continued daily. Should fibrous ankylosis result, mechanical therapeutics and diathermy are frequently of value.

TRACTION.

Direct skeletal traction, preferably of the Pearson ice-tong type, is far superior to indirect. The ice tongs should be properly applied and the weights gradually increased and inspected daily. They should usually be removed in four or five weeks. Their use permits of early mobilization and, if properly applied, will prevent many displacements and deformities, thereby making unnecessary the use of internal fixation and foreign bodies.

THE CARREL-DAKIN TECHNIQUE.

The controversy over the merits of the Carrel method of treatment of wounds has not as yet abated. One frequently hears adverse criticism from those who have little understanding or experience with this method. Those who have used it as advised by Carrel have found it to be most valuable in the treatment of wounds and open fractures. It has enabled us to maintain a pus-free surface. In those cases which are infected on admission, we are now able to eliminate frank pus in from five to ten days, provided it is possible to reach the infected foci, first by surgical procedure and then supplementing this by chemical sterilization. Unfortunately the basic principles of the Carrel method are not generally understood and are not properly practised, and where attempts are made to modify the method to conform to the personal theories of the surgeon, satisfactory results are not secured, and the method is condemned. The success of the Carrel method is dependent upon sound surgical practice, including débridement, and it was never intended and no claims were ever made that it was a cure-all. With more attention to detail and the practice of common-sense surgical principles, better results will be secured. The day of prolonged and profuse suppuration is over and those who still practise methods which are obsolete merely prolong the convalescence of the patient and increase the extent of the disability. If wounds and compound fractures are properly débrided and chemically sterilized with non-caustic hypochlorite, infections would seldom occur. Following chemical

sterilization, should more rapid cicatrization be desired, the application of aluminum potassium nitrate, 13 to 26 grs. to the ounce, incorporated in a poultice of sterilized oats, brings about a more active hyperemia, with a marked tendency toward rapid cicatrization. This method, when used where it is indicated, has been a valuable aid in the closing of sinuses.

SODIUM HYPOCHLORITE SOLUTIONS.

Dakin solution is usually made from bleaching lime and liquid chlorine. As made from these chemicals, it is more or less caustic (alkaline) and is somewhat unstable. Sodium hypochlorite in concentrate is now available for use and can be diluted as per requirements. We have been using an isotonic saline solution with sodium hypochlorite 0.45, and found this to be most satisfactory and superior to the solution formerly made from bleaching lime and liquid chlorine. The ideal antiseptic is yet to be found. A great step in advance, however, is the use of the non-caustic hypochlorite in combination with an isotonic saline. Tr. of iodine, mercury, the silver salts, and the carbolic group coagulate the proteins. These coagulants serve as a culture medium for the growth of bacteria. They are of little or no value in the treatment of wounds or infections and inhibit or destroy nature's efforts at repair. The iodine craze and its indiscriminate use by the profession and laity has given a false sense of security which was never justifiable or based on scientific facts.

MOBILIZATION.

Absolute rest was generally accepted and practised in the treatment of fractures and joint injuries. The Championnière and Willems methods of mobilization have completely upset one of our pet theories and revolutionized the treatment of fractures, infections, and joint injuries. Active mobilization should be started within the first few days and continued daily. The Thomas splint with Pearson flexion attachment is preferable to plaster, as they permit more readily of early mobilization. Consistent with safety, a properly fitting ambulatory brace should be applied and the patient encouraged to walk and use the extremity at the earliest possible date. Prolonged immobilization increases the tendency to ankylosis, extends the convalescence, and increases disability. Early mobilization will reduce the period of convalescence from fifteen to fifty per cent. There are some who maintain that we have gained little new knowledge

in the World War: they can see nothing new and tenaciously cling to methods of the past. Those of us who have accepted and practiced mechanical and chemical sterilization of wounds, early mobilization and the use of Thomas splints, together with proper ambulatory (walking caliper braces), have materially reduced the disability, lowered mortality, and shortened the period of convalescence of our patients. The treatment of joint wounds and fractures of the femur has been revolutionized during the past seven years. The profession is rather reluctant to put in practice progressive ideas. Many hospitals are sadly deficient in armamentarium. Efforts toward elastic standardization should do much to bring about general improvement and results. Conflicting opinions and practices should be compromised with the hope of clarifying the present chaotic conditions.

REPORT OF ONE HUNDRED AND NINETY FRACTURES OF THE FEMUR.*

BY ARTHUR M. SHIPLEY, M.D.

In looking over the literature having to do with fractures of the femur, one is struck by the absence of any standardization in the treatment of these injuries. Equally good men take entirely opposite views, and the reason for this is not far to seek. We are inclined to look upon fractures in the different parts of the femur as one and the same injury. Almost every different fracture presents a different problem, and these problems are met by different men in different ways.

Let us run over the classification briefly. Fractures of the femur are divided into:

1. Fracture of the head.
2. Epiphyseal separation of the head.
3. Subcapital fracture of the neck.
4. Intertrochanteric fracture.
5. Sub-trochanteric fracture.
6. Fracture of the great trochanter.
   Fracture of the lesser trochanter.
7. Fracture of the upper third.
8. Fracture of the middle third.
9. Fracture of the lower third.
10. Supracondyloid fracture.
11. Epiphyseal separation of the lower end.
12. Articular fractures into the knee joint.

Then again these fractures may be transverse, oblique, spiral, comminuted, multiple, pathologic, or complicated.

The age factor plays a very considerable part. The problem in children is for many reasons a very different one from that of old age.

Roughly speaking, the methods of treatment fall into a number of groups:

1. Buck's extension and straight side splint. I was brought up on this method, and within certain limits it is still a good method.

* Read before the American Orthopedic Association, June, 1923.

2. Extension and counter-extension, with the use of the Balkan frame, or some other overhead support, with or without the use of ice tongs or the Steinmann pin.

3. The reduction of the fracture by means of the Hawley table and immobilization in plaster of Paris.

4. The open method.

The second and fourth methods are widely varied in detail. I shall not attempt to discuss these various methods, but shall confine myself to a report of the treatment of fractured femurs in the University Hospital Clinic.

In the last five years, there have been 190 fractures of the femur. Fifty-five of these were of the neck, of which 28 were sub-capital and 27 intertrochanteric. Twenty-two were of the upper third; 87 were of the middle third; 26 of the lower third. Fifty of these were in patients under ten years of age; 68 were between ten and fifty; 72 were over fifty. Practically all of the neck fractures fall in this last group. As to treatment, 138 were treated by the closed method; 52 by the open method. Of these 52, treated by the open method, the Lane plate was used in 22. In 8 the ends of the bones were exposed and brought end to end, but no internal splint of any sort was used. In 12 the ends of the bones were fastened with silver wire. Many of these were in children, where the bones were small and the wire held the fragments in position. In one, a badly comminuted fracture, both a plate and band were used. A bone plate was used in one fracture of the shaft, and bone pegs were used in three fractures of the shaft. A beef-bone peg was used in six fractures of the neck.

In my experience, the Sherman plate is better than the Lane plate for two reasons: the Sherman plate is not so apt to break, and the Sherman screws hold much better than the old ones.

We are almost entirely plaster men in this clinic. This report does not include any compound fractures.

In sub-capital or intra-capsular fractures of the neck of the femur, I believe that the ideal thing is an open operation, with the use of a bone peg driven through the trochanter major into the neck. The extremity is then immobilized in plaster, with the leg strongly abducted, inverted, and lifted forward during the application of plaster. In these fractures, there is a three-fold displacement. The thigh is adducted, rotated out, and displaced backwards. Personally, I am in the habit of placing these bone pegs by using two incisions. The first incision is made over the neck of the femur, parallel and to the mesial side of the tensor vaginæ femoris

muscle, the common incision that is made for resection of the head of the femur. This keeps one well away from the femoral vessels, and usually only one vessel of considerable size is encountered—the upper perforating branch of the profunda femoris. After exposing the neck, a second incision is made just over the trochanter major, and a hole is then drilled through the trochanter major into the neck and head, and the bone peg introduced. I have been using beef-bone pegs. Unfortunately, relatively few cases of fracture of the neck of the femur are good enough operative risks for this procedure. Our feeling is that the next best procedure is immobilization in plaster in the Whitman position. There are many patients who will not stand even this amount of definitive treatment.

Intertrochanteric fractures, in our experience, give a very fair prospect of union. Here again, however, the deformity is adduction of the injured thigh, outward rotation, and backward displacement of the lower fragment. We have been treating these injuries by immobilization in plaster, with the Hawley table, in extension, strong abduction, and internal rotation.

In fractures just below the trochanters, the upper fragment is usually flexed and rotated, and the problem is to bring the lower fragment in the position of the upper. These fractures, in our experience, are often comminuted, and, for that reason, do not lend themselves very well to the open method of treatment. We have been attempting to treat them by placing the leg in such position as to bring the lower fragment in line with the upper. What is needed here is a fracture table, in which extension can be obtained in the different degrees of flexion. Such a table was devised by Dr. Downey of Gainesville, Ga., and reported as long ago as 1909.

In fractures of the middle of the shaft, the problem is also a varied one. In our experience, it is almost impossible to get the fragments in a short oblique fracture in good position. If the fragments in a transverse fracture can be brought end to end, it is not difficult to maintain them so, but a certain percentage of these transverse fractures have a pad of torn muscle interposed between the fragments, which makes it impossible to get the end of the bones to engage, so that in short oblique and transverse fractures of the femur, I believe the open operation offers the best method of treatment in the majority of cases. In long oblique and spiral fractures, it is possible in the majority of cases to get sufficiently good position without open operation.

In supra-condyloid fractures, a difficult problem is always met. The lower fragment will invariably be displaced backward by the pull of the gastrocnemius muscle. It is in this fracture that I believe extension and

counter-extension, with the Steinmann pin, Balkan frame, and flexion of the leg on the thigh, offer the best method of treatment.

In articular fractures, which include the Y and T fractures into the joint, we have found that in most cases reduction can be brought about by molding the displaced fragments with the hand and immobilizing in plaster.

If this cannot be done, the fragments should be replaced by open arthrotomy of the knee joint, with the use of bone pegs to hold the fragments in position, if they do not remain there of their own accord after replacement.

Fifty of these fractures were in children under ten years of age. We have invariably treated fractures of the femur in children by immobilization in plaster. The plaster extends from well above the costal margin to the base of the toes of the injured leg, with the leg in abduction and flexion. The uninjured leg is immobilized as far down as the knee, also in abduction. It is a comparatively easy matter to get sufficient extension in children while the cast is being applied. Such a cast makes it easy to handle them; they can be carried from place to place, and, if a wide opening be left about the genitalia, comparatively little soiling is encountered.

DISCUSSION ON PAPERS OF DRS. SHERMAN AND SHIPLEY.

Dr. Clarence Starr, Toronto, Ont.: I think the Society is to be congratulated on having the opportunity of hearing the presentation of this subject by two masters in the art. I just want to add a few words and call attention to a few facts. Some of the statements made by Dr. Sherman might lead to the belief that he advocated his technique in all compound fractures. He will agree, I am quite sure, that the bulk of civilian fractures, compound in type, are not of the character he showed on the screen and his talk must apply to that type. I am sure it is his experience, as it is ours, that the bulk of compound fractures are produced by the bone sticking through the skin, and the bulk of those require no *débridement*, no Carrel-Dakin solution, and no irrigation of any kind; in fact, they require a large amount of leaving alone, treating them as simple fractures such as Dr. Shipley has described. I feel this very strongly from the large number of compound fractures that come to us directly on injury, in which we have not seen a single infection. On the other hand, we have seen a large number of infections in cases treated outside by men who feared infection and attempting to prevent it by resection and other means succeeded only in producing it. Leaving out that group of cases which I feel should not be included in this discussion, and which we treat as I have suggested, we come to the other type of severely injured patients who have much greater danger of infection. I try to impress on my students that the man who sees the case first must assume the responsibility for the subsequent condition of the wound, as he has a much better opportunity to prevent infection than the surgeon who sees the case subsequently. If the wound afterwards becomes septic, it is treated as an infected wound.

The cases in civilian life that have injuries such as Dr. Sherman has pictured are from machinery injuries, and the bulk of them which have come to *débridement* for these injuries will probably require amputation later in a large percentage of the cases, the vessels and nerves being destroyed with the other soft tissues. It has not been my experience in civilian life that *débridement* is frequently necessary. You will recall that the reason for *débridement* in the majority of cases was infection; not the destruction of tissue so much as the fact that the destruction of tissue made a lodging place for the gas bacillus, and the gas bacillus infection which we had was responsible for the high mortality in those cases. So *débridement* in the civilian type of cases must be limited to a comparatively small number of the entire group, and should be surely and carefully done.

The question of internal splinting or fixation of any sort is, as Dr. Sherman suggested, a simple matter of fixation. The presence of infection does not usually mean that we will not get union, because it is pretty well demonstrated that septic fractures will unite if given a reasonable chance. The whole factor of fixation by splints, wires, clamps, or what not is simply to keep the fragments approximated sufficiently accurately that union may take place, because in septic wounds, as in simple fractures, the cause of non-union is lack of apposition. To the question of fixation by splint, Lane plate, or what not, if taken out at the end of four or five weeks, no one can take any exception, unless in the severely infected ones where it may open up a larger field for infection. We have found that simple fixation by an absorbable type of material such as kangaroo tendon, and at the same time a Thomas splint and traction properly applied, give excellent results.

Now Dr. Sherman's contention that war technique should apply to infection out of the war is absurd to a degree, for I feel sure that there was a large percentage of stuff learned in the war that we must forget because it does not apply in civilian surgery. One thing we did learn that has received entire commendation is the use of the Thomas splint. I think if we learned nothing else it is well worth while to take that lesson to heart in the question of fixation of fractures. I personally feel that with the aid of the Thomas splint the majority of fractures can be fixed sufficiently by absorbable material to get good approximation. I am free to confess that the longer I practise and the longer I fix fractures, the less steel I use. I am perfectly sure that a very large percentage of our cases of non-union in Army service was due to the use of plates.

I want to emphasize what Dr. Sherman mentioned, that fragments which are entirely separated should be removed. I would give those that are not entirely separated a chance, because they can be removed subsequently. I am quite sure that some of the fragments removed still have enough attachment for their vitality and can be returned, so for the most part I would not remove a quantity of bone.

The question of infection: I did not quite gather whether Dr. Sherman felt that the use of the Carrel-Dakin solution was advised in cases which were not infected or only in the infected ones. If not infected, it seems to me that I would hesitate to introduce any irrigation in a wound which is not previously infected.

The question of traction: We have very rarely used the ice-tong type of traction. We use the Thomas splint with the joint at the knee, permitting movement at the knee and ankle. In some cases in the lower end of the femur the ice-tongs have a very definite place.

The question of the time of splinting, I feel that Dr. Sherman, in talking to a

group of intelligent gentlemen such as is represented by the American Orthopedic Association, does not need to emphasize the time, but in talking to students it is absolutely necessary to emphasize the question of time. I have seen very good reduction and very good primary position absolutely destroyed by too early removal of the splint. I do not believe that six weeks is long enough to splint a femur whether it is simple or compound, although we have a number of persons who advocate that. I think Dr. Sherman should emphasize the question of time.

In regard to Dr. Shipley's method there is a rather interesting coincidence. For a number of years I had a large service at the Children's Hospital and I advocated treatment by plaster splints in fracture of the femur. I advocated it so strongly that I was perfectly convinced that anyone who did not use it was missing the best type of treatment. There was one type of fracture which we felt we did not control and that was the transverse fracture. We felt there were a large number of cases in which we could get a perfect end-to-end union and there were a fair number in which we could get apposition by irregularities in the bone ends which would lock. We had a perfect line, good fixation, no abnormality, and we got union without difficulty. There was another group that we could succeed in reducing, particularly those in the upper third, and whether you put them straight or bent there was a certain percentage in which the pull of the psoas muscle would jar them out of position. We were so distressed by these cases that we made it a habit to make a radiogram at the end of six days after primary reduction, which was perfectly controlled by radiogram, to see whether it was out of place. If it was out of place we knew it would not stay a second time and we opened up the thigh and put on a fixation splint. The usual type of fixation splint, the steel splint, and steel screw were discarded and we used altogether, due to the ingenuity of my associate, Dr. Gallie, the bone screw. This bone screw was made on the same principle as a steel screw and we are fairly satisfied with the result. I am quite sure that my successor in the Department will say he is getting equally good results without plaster and with the Thomas splint. Since my transfer to the General Hospital my work is entirely with adults and I find that plaster fixation is difficult and the Thomas splint is so satisfactory that we are using it practically all the time. Fractures of the middle of the shaft in adults can be brought to length and alignment by this means. We found that instead of our straight traction being confined to fractures in the middle of the shaft, we can control those in the upper and lower third. In those that go down to the condyles, there is no question that bending the thigh and bending the knee relax the muscles and make it easier to get it in position. Those cases which have aponeurosis and muscle interposing have to be opened up. Even in those which have to have a large internal splint we rely on the Thomas splint subsequently. Our experience is that in practically no department of surgery do we find steel plate and operative procedures used in more than 5 per cent. of cases in which they were used five years ago. The results have been more satisfactory in that union has been more rapid and function has been established earlier and altogether I am very well satisfied with the method of treatment along that line.

Dr. Willis C. Campbell, Memphis, Tenn.: The subject of fractures of the femur is too broad to discuss all phases. Recently I have had an opportunity of compiling, from our private records, 226 cases of fracture of the neck of the femur,

including trochanteric fractures and fractures in and about the neck. Dr. Speed, my associate, has reviewed 173 fractures of the shaft, making a total of 396. Five years ago, before the American Medical Association, I stated that 90 per cent. of the central or intracapsular fractures of the neck of the femur healed by apparently bony union, with proper treatment. Since that time this has been frequently refuted and the statement has been made that rarely does bony union occur in this location.

In order to determine the actual results obtained we made a survey of about 21 central or intracapsular fractures, making radiograms and keeping close observation, no conclusions being accepted by letter. The result has shown 90 per cent. solid bony union, a detailed report of which will be published at a later time.

Our results in fractures of the shaft, by simple reduction, have been so satisfactory that open reduction in fresh fractures has been rarely undertaken. For instance, rarely can reduction be accomplished by forcible traction in transverse fractures, but by acute angulation the ends can usually be approximated and retained in plaster cast until union is solid.

Dr. Edwin W. Ryerson, Chicago, Ill.: I want to speak of one point only, in the treatment of fracture of the neck of the femur. We should not speak of putting a peg, or screw, or nail "through the trochanter." It gives the wrong impression and leads to improper surgery. If it is deemed advisable to peg or nail a fractured neck of the femur, then the drill-hole must be started well below the trochanter and must be inclined upward and inward at an angle corresponding to the direction of the neck, usually about 135 degrees. If this is done, the pull of the muscles will force the neck and head strongly together, and even a small peg will stand the strain.

If, however, the peg is put in too high, actually through the trochanter, the direction will be nearly horizontal or at right angles to the shaft, and the muscle-pull will tend to distract the head and neck, and even a strong peg may be broken. Dr. Shipley showed an X-ray of a little bit of a peg going horizontally through a trochanter, and of course it broke, because it was put in the wrong place and in the wrong direction. Dr. Baer also spoke of putting pegs through the trochanter. I think none of us should use this form of expression in the future.

Dr. H. Winnett Orr, Lincoln, Nebr.: Dr. Sherman tells us that *if débridement* is well done and if chemical sterilization has been accomplished the opportunity for secondary suture might present itself. He did not mention two fundamental points which it seems to me are necessary if we are going to get results. These two points are drainage and immobilization. At any rate he did not mention them in that connection. It has always seemed to me if there were chemical sterilization and complete immobilization of fractures in the neighborhood of joints the result would be good. But these two things must go together. The lesson we learned from the war, if we learned any, was that the Carrel-Dakin treatment and the Willems treatment, in so far as they interfered with immobilization, were a mistake. That good results were often obtained is true, but these methods are not for general use without a due regard for other things.

DR. A. H. FREIBERG, Cincinnati, O.: I do not rise to discuss these very admirable papers because I have anything notable to contribute, but to call attention to one thing: that is, the likelihood of our putting cases into groups without sufficient distinction. I feel as a result of a rather long experience that fracture of the femur in a child is one thing and fracture of the femur looking very like it in an adult is an entirely different thing. The mechanical conditions which we bring to bear on these cases operate very differently in these two classes. You can accomplish very easily in a child a result which you cannot accomplish at all in an adult with the same kind of mechanical appliance. The child recovers with an admirable result from a kind of fracture which appears to be the same in an adult but in whom we cannot accomplish as much with this simple treatment as we have been able to get by with in the child.

The Thomas splint is a very admirable splint. In my opinion the Thomas splint is an entirely different proposition used on a man from what it is when you put it on a woman. A degree of traction can be tolerated very much better in a man than we can possibly bring to bear even with efficient nursing care in a woman; at least that has been my experience.

When we come to study compound fractures I believe we have a very different situation to deal with on different occasions. The situation in war wounds was very different from what it usually is in civilian life and I dare say that Dr. Sherman with his ordinary work comes nearer to seeing fractures similar to war wounds than most of us do. It makes a difference whether the damage to the soft parts takes place from within or from without, and this same thing happens under different circumstances. I believe we should be somewhat careful in accepting things that are said in too dogmatic a way, even though all of them are probably true under certain circumstances. One of the speakers has said, "each case is a law unto itself;" perhaps he did not even realize how truly he was speaking.

DR. JOHN L. PORTER, Chicago, Ill.: I hope Dr. Shipley will tell us why he so studiously avoided the use of the autogenous bone splint in fractures of the femur that are not infected. I am sure a large number of us are using fewer metal splints and Lane plates every year. I do not think I have used one in the last four years and I am very sure that I have better results with bone splints in fractures of the femur, which cannot be held in proper alignment, than I ever had with metal bands.

DR. G. D. MARSHALL, Kokomo, Ind.: I want to mention one thing in the treatment of fractures of the femur. I visited the Boston meeting of the American Orthopedic Association and saw Dr. Bradford apply an abduction splint for a fracture of the neck of the femur. I believe I have been fairly repaid for the trip to Boston after seeing this splint. The advantage it has over the Thomas splint is that it prevents rotation. Many of these short spiral fractures are not held in apposition because there is a certain amount of rotation. I do not believe this Bradford splint is recognized as much as it should be. It has many advantages besides preventing rotation, that is, it affords a larger bearing surface for the counter traction on the hip than you get with the ordinary splint. You get it under both tuberosities of the ischium and it is held there.

DR. JOHN N. BASSIN, Newark, N. J.: I have heard a great deal about fractures of the neck of the femur and am extremely interested in not having heard mentioned another method of treatment for those fractures called intracapsular where every method apparently fails because of the constitutional condition and age of the patient, especially about the age of fifty. I heard it mentioned here that about 50 per cent. of fractures of the neck of the femur do not give final good results: that is, there is a fibrous union or a non-union. These are usually relegated to the scrap heap. In all events the old folks either have to wear a hip brace, use crutches indefinitely, or they simply go on to the old people's home or the poorhouse. The industrial cases of this type present a big economic problem. Not over a year ago I heard Lorenz describe his open method of hip operation. I am speaking for the principle, as it occurred to me that in some cases where everything else had failed this method might be tried. I did try it on several cases of fracture of the neck of the femur with a complicating multiple fracture of the pelvis and then on a few other cases where I had a fracture straight through the neck and union did not take place in spite of the Whitman method. I modified the operation by not only doing an osteotomy under the trochanter major and minor but also severing the insertions of the gluteus medius and minimus, thereby shortening the bone gap which otherwise takes from three to four months to fill in. So far in the few cases operated on we had very good results with bone union and more than 50 per cent. excursion of the joint.

DR. WILLIAM O'NEILL SHERMAN, Pittsburgh, Pa. (closing his part of the discussion): In reply to Dr. Starr's discussion I will say, the questions to which he takes exceptions were definitely covered in the paper. The environment in which the fracture occurs is very often an important factor in determining the treatment. Those of us who are identified with the large industrials, railroads, mines, etc., see the same type of trauma as seen in the World War, and the compound fractures are very much more extensive than those occurring in the lighter industrial communities where the gross tonnage is much less.

There are certain principles which are absolutely necessary if lives are to be saved and amputations prevented. The Carrell-Dakin solution is used as a prophylactic just as antitoxin is used. If used promptly, infection will rarely occur.

After the plates have been allowed to remain for four or five weeks, they have served their purpose and can be safely removed. Vanadium steel plates are preferred because of the maximum physical properties contained therein. The breaking of plates and pulling out of screws is a very unpleasant complication. Wires and beef bones are not sufficiently strong to warrant their use. One would not relish being confronted in a Court by a case in which beef bone plates had been used.

The convalescent period has been materially reduced by proper operative treatment and early passive motion. Many surgeons have been content with three-fourths of an inch shortening in fractures of the femur. Such results are hardly a credit to the profession. It must be remembered that the majority of individuals who sustain fractures of this kind are wage earners, and a reasonable approximate end-to-end result is necessary to permit the patient to return to his former occupation. Operative procedure is absolutely contra-indicated unless one has the necessary facilities and surgical armamentarium.

Following open operation, mobilization is started within the first three or four days. Many of these patients are now resuming their work in half the former time. If a Board of Supervision were created to review the end results in cases of fractured femurs, a great many would be found in vicious positions, with great impairment of function. The treatment of this type of fracture has now advanced to such a stage that one can say that such conditions should not exist. We have been using the Thomas splints for the past five years, to the exclusion of plaster in the treatment of femur fractures.

I cannot agree with Dr. Orr's statement with reference to chemical sterilization and immobilization. Lack of knowledge as to the chemical action of sodium hypochlorite, together with its instability and caustic action when not properly made, and poor technique, have interfered to a great extent with its general acceptance as a prophylactic and its use in the treatment of infection. One can say positively that infection should not exist today in compound fractures, providing the case is given early and proper treatment.

Dr. A. M. Shipley, Baltimore, Md. (closing his part of the discussion): As to inlay grafts, medullary grafts, bone pegs, and bone plates in fractures of the femur, our experience has been that they break. Not all of them break, but every once in a while one breaks. Bone is brittle. In inserting them, if there is the slightest bit of motion, the inlay graft is broken. I do not believe an intramedullary graft should be used in a fracture, because it promotes non-union. The inlay graft will snap. We take out Lane plates and Sherman plates. I always remove them from the tibia, but not from the femur, if they are well covered by muscle, and I think in the majority of cases you never hear from them again.

Dr. Freiberg spoke about the difference between children and adults. There is a tremendous difference. There were fifty children in this series. In treating fractured femurs in small children, one is strong enough to overcome the muscular pull and you can handle them. If we were giants we might be able to handle adults as well as we do children. It is the lack of strength on the part of the surgeon that prevents his unlocking fractures in adults. Only four of these fifty children were treated by open operation, the other forty-six were treated by closed reduction.

CLAW FOOT—ITS TREATMENT.

BY F. WILSON STUART, BIRMINGHAM, ENGLAND.*

In presenting for your consideration some views on the treatment of Claw Foot my remarks will be limited to those cases requiring operative interference. As much of what I have to say is already well known to you, the methods employed will be discussed from the standpoint of degrees of deformity and an endeavour made to make clear their standardisation.

The aim in treating this condition is to reduce the abnormally high arch so as to enable the patient to walk on his natural weight-bearing points and be free from pain. To obtain such a result two methods are available, each to be employed according to the degree of the deformity.

One depends for success on division of contracted soft structures, combined with wrenching, the other on removal of bone. Before deciding on which method to adopt a correct appreciation of the architecture of the foot is necessary.

Examination of a series of cases shows that there are two definite types of the deformity, one in which the first metatarsal head can be replaced, the other in which it cannot.

In the earlier stages of the first there is not much evident increase in the arch, but the plantar fascia is contracted so that it comes easily into view under the skin of the sole when the two pillars of the arch are separated manually; later the increase in the arch is so pronounced that it appears to be almost angular; replacement of the first metatarsal head does not completely obliterate this angularity at the medio-tarsal joint; the plantar fascia no longer stands out when the pillars of the arch are separated, owing to a general contracture of all the soft structures having taken place and to their being pulled up towards the keystone of the arch as it increases in height. Varus deformity and inversion of the heel frequently appear at this stage and give rise to a good deal of pain over the fifth metatarsal head.

The condition of the Achilles tendon is important. It may be normal, contracted, or lengthened. If the latter, which is sometimes due to a

* Paper read at the Annual Meeting of the British Orthopaedic Association, Birmingham, October, 1923.

previous tenotomy, it allows the heel to drop and a consequent increase in the height of the arch takes place.

In the earlier cases then, simple division of the plantar fascia and wrenching will be sufficient to reduce the deformity. In the later stage it is necessary to divide all structures down to bone, and for this purpose Steindler's operation or some modification of it may be employed. The modification I use is performed as follows:

FIG. 1.

Radiogram showing severe claw foot deformity with the amount of bone to be removed from the midtarsal region.

The incision, 1½ to 2 inches long, is made on the inner side of the foot, with its centre opposite the inner tubercle of the os calcis. The skin and subcutaneous fat are separated from the plantar fascia by dissection and a periosteum elevator inserted into the wound until its curved end is felt to pass over the outer border of the foot; the plantar fascia, muscles, and periosteum are now divided, the former well back on the tuberosity of the os calcis, the latter close to its tubercles; the abductor hallucis is also divided at its origin from the internal lateral ligament. All the divided structures are now raised from the bone as far forward as the calcaneo-cuboid joint and the deformity corrected by the hand or wrench. The wound is sutured either before or after wrenching.

I have used this operation with satisfactory results, but have obtained equally satisfactory ones by subcutaneous division of all soft structures down to bone.

The chief immediate advantage of the Steindler method lies in that it is easier to wrench the foot afterwards; this is due to the separation of

FIG. 2.
The foot encased in plaster of Paris with a window over the incision

the structures divided from the skin and subcutaneous tissues and from bone which is not possible by dividing with a tenotome.

Another advantage is said to be in the new forward attachments the muscles and fascia obtain on the os calcis. Allowing this, I do not think it will prevent the deformity recurring, for these same structures still extend across the mid-tarsal joint.

A word on wrenching. There is a tendency to screw the wrench too tightly on the foot, with the result that serious injury sometimes occurs to the tarsal bones. The wrench is sufficiently tight when the upper bar lies across the neck of the astragalus and the lower behind the first metatarsal head and it can be maintained there by the left hand placed on the upper bar.

FIG. 3.
Right claw foot before operation.

Downward pressure is made on the upper bar and upward at the end of the handle. Having got the anterior part of the foot well dorsiflexed at the medio-tarsal joint, it is kept there for a short time to allow complete stretching of the soft parts. Should the Achilles tendon be long, allowing the heel to come down to such an extent that the force exerted in wrenching is entirely lost in dorsiflexing the ankle joint, an assistant must counter-extend at the heel.

Over-correction should be aimed at, as the deformity tends to recur, and if the toes are acutely flexed and the flexor tendons prevent them being extended, the latter should be tenotomised while the foot is held in the over-corrected position by the wrench.

Some surgeons elongate the Achilles tendon at this stage, a proceeding

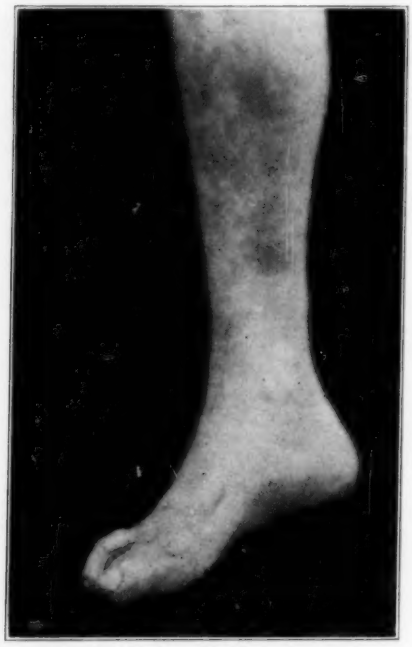

FIG. 4.
Result, after the operation described in the text.

which cannot be too strongly condemned, for it is practically undoing straightaway what has been accomplished and frequently ends in complete relapse.

We must continually bear in mind that we are dealing primarily with contracted sole structures and not with contracted calf structures. Personally I find that the stretching which the tendon gets at the time of wrenching is sufficient for the moment and any further lengthening it

may require will take place as the patient gets about in an altered boot and in the gymnasium. It should be lengthened as a last resource and only when absolutely necessary.

The after treatment consists in allowing the patient to walk in plaster as soon as pain has entirely disappeared; in Steindler's operation when the wound is healed. This is continued for at least two months, then an ordinary boot with bar across the tread is worn and a removable plaster at night.

FIG. 5.
Radiogram of the corrected foot (see Fig. 1) showing sound arthrodesis.

Attendance for massage, re-education of muscles, and stretching of plantar structures is essential if a really good result is to be obtained.

In the second type of claw foot, *i.e.*, where the first metatarsal head cannot be replaced, attempts to do it result in movement at the ankle joint, the heads of the metatarsals appear as a convex projection under the skin of the tread, there is complete dorsiflexion of the smaller toes and any

attempt to flex the metatarso-phalangeal joints produces much pain and fails, something more radical than that described is necessary.

The operation I perform for this condition is modelled on the procedure originally adopted by Mr. Naughton Dunn for the stabilization of the flail foot.

It consists in resecting the mediotarsal joint, the whole scaphoid being removed, in transplanting the tendon of the extensor proprius hallucis to the first metatarsal head and arthrodesing the first interphalangeal joint.

The exposure is through a curved incision on the outer side of the ankle and foot with its centre opposite the neck of the astragalus, and goes down to bone, and a L-shaped incision over the first metatarsal head and interphalangeal joint. The dorsal tissues including skin, vessels, nerves, and tendons are now raised in a flap by dissecting across the foot until its inner border is reached.

The mediotarsal and scapho-cuneiform joints are opened by dividing the ligaments connecting them on their dorsal and inner aspects. The scaphoid can now be removed complete by inserting a gouge under it, which divides the plantar ligaments. (Fig. 1.)

The head of the astragalus and cuneiform bones are stripped of their cartilaginous surfaces along with a thin shell of bone, preserving their natural contour as far as possible. The calcaneo-cuboid joint is similarly dealt with, sufficient bone being removed to allow of the distal part of the foot being placed in a correct position and firm contact established between the raw bony surfaces. If the plantar fascia still prevents proper alignment being obtained it should be divided at this stage.

An assistant now holds the foot in its new position while the cartilage is removed from the adjacent surfaces of the interphalangeal joint and the tendon of the long extensor of the great toe passed through a tunnel made for it in the first metatarsal head.

The wounds are sutured, a small counter opening being made below the angle of the larger for drainage purposes.

As the operation is performed with a tourniquet it is well to remove it before closing the wounds so that hæmorrhage, if excessive, can be dealt with. As a rule this does not occur, but if it proves troublesome then injection of saline at a temperature of 120° F. will stop it at once.

Dressings are applied and the foot encased in plaster of Paris, care being taken that the toes are perfectly straight. Lids are cut out from the plaster cast over the sites of both incisions and a gauze dressing soaked in spirit applied over the original incision. Incisions are made on either

side through the whole thickness of the plaster to within a short distance of its extremities and the ankle joint. This not only allows for swelling, but it makes removal easy should necessity arise.

About the seventeenth day the plaster is changed to allow of moulding of the foot, if necessary. If left longer, it is impossible to mould the foot without an anæsthetic. Moulding is seldom required and should not be, if a proper position has been obtained at the operation.

Walking in plaster is permitted at the end of six weeks and this is continued for six months, when an ordinary boot with double iron may be worn.

Massage and re-education of muscles, mobilization of ankle and toe joints is now begun, and by the end of a year the leg should be sufficiently strong to allow of the iron being discarded.

MECHANISM OF THE NORMAL FOOT AND OF FLAT FOOT.

PART I.

BY DUDLEY J. MORTON, M.D., MILFORD, CONN.

INTRODUCTION.

In the previous paper* the origin of the human type of foot was considered and the manner in which its peculiar characteristics were evolved. The purpose of the present writing is to analyze the physiologic function of the modern foot, noting the earlier stages of functional mechanism as demonstrated by the foot of the gorilla and of pre-historic Neanderthal man, and then to apply the information gained by these investigations to a more practical end—a study of disordered function.

From the viewpoint of orthopaedic surgery, the results of this research into the evolution of foot structure and its function appear to have more than ordinary value. Some new and helpful thoughts have been furnished; one finding in particular—the os calcis as a means of regulating "balance" in the distribution of body-weight—seems to justify recognition as a distinct therapeutic principle, so positively does the influence of its position bear upon the normal performance of foot function.

The mechanism of the foot would be a very simple problem if the foot structure consisted of a single bone, but because of the many bony segments that enter into its makeup, and the peculiar distribution of body-weight and muscular force into those various segments, the mechanism becomes a somewhat complicated one.

Each segment of the foot has a particular share in the complete function, just as each chamber of the heart has in the function of that organ. Looking upon the foot as a structure into which body-weight enters near the heel, to be eventually transmitted to the ground or propelled forward, is on a par with considering the heart an organ that receives blood into its right auricle and pumps it into the aorta by the left ventricle—a perfectly correct interpretation, but one that does not constitute a working knowledge sufficient to treat cardiac disorders very intelligently!

* Evolution of the Longitudinal Arch of the Human Foot. J. Bone and Joint Surg., Vol. VI, No. 1. January, 1924, pages 56–90.

The necessity of a clear conception of the foot's mechanics for intelligent corrective work, especially operative, has been most strongly indicated in these studies. Any operation upon a deformed foot that is based only upon a consideration of external appearance is like a drug that is prescribed for a heart case after a mere feel of the pulse.

Only through a clear understanding of how the function of the foot is properly performed and how each portion of the foot is involved in that function can a correct interpretation of its disorders be made and the disorders most efficiently remedied. Abnormal contour of the foot naturally facilitates a diagnosis, but treatment must be devised primarily upon a comprehensive rearrangement or correction of the inside mechanism in a strictly scientific manner, and not be a procedure aimed only toward cosmetic effect.

Preservation or restoration of normal foot mechanics will insure normal contour; work that fails to correct an improper distribution of bodyweight through the foot will inevitably lead to increasing and more extensive deformity.

THE FOOT AS A WEIGHT-SUPPORTING STRUCTURE.

The bones alone do *not* comprise the actual base (*i.e.* the supporting structure), but it is composed of the *bones plus their plantar ligaments.* While the arched contour of the human foot is undoubtedly the direct product of active leverage, similar properties are also required in an arched structure for passive weight-bearing. They are: (1) the quality of transmitting weight without change of form and (2) resistance to tensile stress extending from one extremity to the other. The bones of the foot alone do not fulfill these requirements and, therefore, could not constitute a lever or a weight-supporting arch, any more than a bag of marbles. The bones possess the hardness by which weight can be transmitted, but the second qualification is met only by the inclusion of the plantar ligaments, as has been brought out in the former paper. A clearer conception of the foot as a functioning structure is gained, therefore, by regarding the plantar ligaments as *flexible continuations of bone trabeculæ which bridge inherited "interruptions"* (joints) *in the bone structure.* The essential combination of bone and ligaments occurred while the arch of the foot was being formed as a product of terrestrial leverage function.

By the same interpretation the plantar ligaments are clearly differentiated from other ligaments of the foot, whose function is not that of weight-bearing, but is merely limited to the inhibition of excess joint movement.

STANCE.

In the act of standing the weight of the body is transmitted to the ground through the foot at three principal points: the heel, and the heads of the first and fifth metatarsal bones (Fig. 1). This tripod arrangement is effected by the heel acting as a posterior pillar to two divergent longitudinal arches, an inner arch HWI which lies practically in a vertical plane coincident with the plane of leverage action, and an outer arch HWV whose plane tilts inward at an angle of about 45 degrees toward the center of weight distribution in the body of the astragalus.

FIG. 1.

Human foot showing lines of structural support. H, heel. W, weight center in astragalus. I, head of first metatarsal. V, head of fifth metatarsal. HWI, — inner longitudinal arch. HWV outer longitudinal arch.

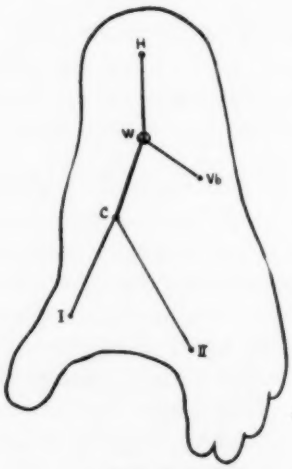

FIG. 2.

Gorilla foot, showing lines of structural support. H, heel. W, weight center. C, inner cuneiform bone. I, head of first metatarsal. II, head of second metatarsal. Vb, base of fifth metatarsal.

This arrangement did not exist in the primitive flexible arboreal foot (Fig. 2). In that structure, as manifested by the anthropoid ape foot, the outer arch was flattened sideways upon the ground and there was no inner longitudinal arch. The abrupt inward slope of the calcaneal

facets deflected the body-weight so strongly against the inner border that the inner cuneiform bone was forced to the ground and became one of the major points of contact. Divergence of the first metatarsal and hallux was necessary in that stage as a means of lateral stability to act against the inward thrust of body-weight. The center of weight distribution descended into the very head of the astragalus, whence body pressure could move only forward through the scaphoid; the outer portion of the foot was consequently almost functionless in so far as support of body-weight was concerned.

Later, when elevation of the inner border developed, extensive alterations in the mechanics of the foot occurred and the outer longitudinal arch became an important, functioning structure. The principal changes referred to are: (1) a remodeling of the os calcis by which the arc of its neck was raised with (2) some degree of outward and upward rotation by which the inward slope of its facets for the astragalus was correspondingly lessened; (3) the cuneiform bone became lifted and ground contact was transferred to the metatarsal heads; (4) the cuboid was raised upon the base of the fifth metatarsal, at the same time elevating the anterior end of the os calcis; (5) weight which had formerly been thrown upon the second metatarsal bone and had led to its notable hypertrophy (as shown by the gorilla foot) was then increasingly assumed by the fifth metatarsal.

While the outer arch is merely an altered position of a pre-existing structure, there is nothing derogatory to its importance in such a comparison of the origin of the two arches; the relative position and contour of the bones along the outer border of the primitive foot were well adapted to furnish a means of stability that was absolutely necessary to the development of the inner arch. The rigidity of the outer structure was essential in order to restrict the freedom of movement permitted by the astragaloscaphoid joint.

The diminished inward slant of the calcaneal facets and the superior position occupied by the astragalus in relation to the other bones of the foot as a result of these changes caused a considerable modification of the distribution of body pressure; body-weight was thrown less strongly inward (thereby lessening the necessity of a wide divergence of the hallux) and the outer half of the foot correspondingly assumed a greater share of the burden.

LOCOMOTION.

The principle of leverage as applied to the foot has already been discussed in a general manner in the previous paper. Here we consider

more fully the distribution of forces and the share which each part of the foot plays in the function of locomotion.

The Fulcrum. There are several reasons for designating the heads of the first and second metatarsal bones as the principal fulcrum for the leverage action of the foot:

1. The heads of the first and second metatarsal bones are more distally located than the others.

2. The greater length and increased development of the human foot is located along the inner border.

3. The most conspicuously developed arch is placed along the inner margin of the foot.

4. The possibility of the characteristically human evolutionary development from a primitive type of foot could have been conditioned only by the major part of the body-weight being borne by the inner portion of the foot.

The frequent interpretation that the line of leverage action in man moves along the central axis of the foot is not substantiated by evidence to be found elsewhere in Nature, because invariably the size of each metatarsal and its digit corresponds to its respective share of function, and that function is always concentrated along the line of leverage.

The heads of the three outer metatarsal bones may be looked upon as the fulcra of auxiliary levers. The function of those bones is quite important when the heel is first raised from the ground, but as the movement continues their heads become lifted so that they are then rendered ineffective.

Power. The major propulsive effort is furnished by the larger calf muscles, gastrocnemius and soleus. Normally their power is exerted directly in the vertical plane of leverage action, in which lie the centers (1) of the lifting power from these calf muscles, (2) the weight center in the astragalus, and (3) the fulcrum.

The bowstring action of the flexors, peroneals, and posterior tibial muscle increases the propulsive force to a considerable degree, but of particular importance is the lateral pressure which they exert at various points on the foot.

Leverage Action of the Foot. Counter-pressure furnished by the lifting pull of the calf muscles throws the body pressure into the forepart of the foot. It becomes divided and follows two channels through the foot structures: (1) along the inner border through the neck of the astragalus

(which may be termed the Scaphoid stream), and (2) through the anterior process of the os calcis (the Cuboid stream) (Figs. 3 and 4).

There is a third course *over* the foot which is taken by that portion of body pressure that has been converted by the propulsive effort into momentum. With the conversion of body-weight into momentum there is a continuously decreasing load of body pressure upon the foot structure.

The bifurcation of the bony channel which causes the separation of the stream of pressure through the foot also causes a division in the propelling force so that one portion curves by a higher arc upon the body of the astragalus, throwing the Scaphoid stream of body pressure into the neck of that bone, and the other portion moves by a lower arc toward the upper surface of the anterior process of the os calcis to act upon the Cuboid stream.

The movement and the effects of the propelling force are largely influenced by the laws of ballistics. With more violent efforts the force is thrown more strongly into the higher channel and body pressure will be more strongly directed along the Scaphoid route and over the top of the foot. In slow locomotion the force tends correspondingly to follow a lower arch and become increased in the outer channel.

The Cuboid Stream. Moving through the cuboid bone there are two courses open to the outer stream of pressure: (a) downward into the base of the fifth metatarsal bone and (b) obliquely upward across the foot toward the base of the first metatarsal by way of the cubo-cuneiform articulation.

As in the original division of pressure, the strength of the force acting upon the pressure-stream determines the direction and proportionate strength of the subdivided stream. In slow movement the lower course (a) is chiefly followed, where the pressure is acted upon by the peroneus brevis muscle and an extension of the influence of the larger calf muscle through plantar ligaments and intrinsic muscles. The fifth metatarsal bone is employed as a lever to raise the pressure into the fourth, through the inter-metatarsal facets between the bases of these bones. In a similar manner the pressure is passed successively by the outer metatarsals inward and upward to the inner side of the foot.

The middle channel (b) is followed by that portion of pressure which is affected by the action of the peroneus longus muscle. This muscle throws the stream more directly toward the inner border of the foot because of its location beneath the cuboid bone and its attachment to the first metatarsal base. When locomotion is rapid, increased counter-pressure extending backward through the outer metatarsal bones produced

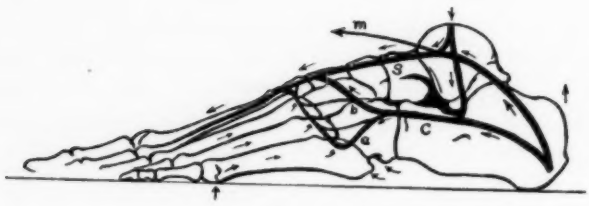

FIG. 3

FIG. 4.

Lateral and dorsal views of human foot, showing movements of force. S, Scaphoid stream. C, Cuboid stream. b, lower and upper channels of Cuboid stream. m, momentum. The small arrows in the three outer metatarsal bones represent counter pressure from the ground.

by the more forceful muscular action, augments the pull of the peroneus longus muscle in directing the entire cuboid stream along the more direct channel (b).

The Scaphoid Stream. That portion of body-weight (S) which follows the inner border of the foot is influenced first by the mild inward slant of the calcaneal facets, and in locomotion also by the direct thrust of the larger calf muscles. Its movement is not directly forward but forward and inward, following the axis of the neck of the astragalus, and off the side of the foot. Meeting the lateral resistance of the flexor digitorum and then of the tibialis posticus muscles, the stream is redirected back upon the foot to unite with the converging Cuboid stream from the outer side of the foot, at about the juncture of the two inner cuneiform bones with the base of the second metatarsal. In slow locomotion and stance the lateral curvature of the Scaphoid stream is quite marked, but in rapid locomotion its arc becomes flattened, following more closely the actual line of leverage of the foot as in the case of the Cuboid stream; the more forceful thrust of the calf muscles not only increases the amount of pressure that travels by the Scaphoid route, but it also causes it to move more directly in a straight line.

Metatarsal Function. The anterior leverage portion of the foot consists of the metatarsal bones. The work performed by the outer ones has already been described; they are the means of reverting the Cuboid stream back upon the inner side of the foot.

After the fusion of the two streams, the pressure follows along the principal line of leverage to the fulcrum, through the shafts of the first and second metatarsal bones. The first metatarsal carries the greater portion of this load, as is indicated by its size. The actual position of the principal line of leverage seems to be logically placed near or in the almost vertical plate which forms the outer aspect of the shaft of the first metatarsal bone.

The Structural "Balance" of the Foot. The foregoing analysis of the distribution of body pressure may have seemed to be unnecessarily detailed, but the distribution of body pressure comprises the most important factor in the function of the foot.

In order that weight be propelled in the direct line of leverage it must be *balanced* upon that line. It is absolutely essential, therefore, that in order to compensate for pressure which is transmitted to the outer side of the foot for the purpose of stability, a *counterbalancing* share of pressure be diverted from the inner side of the line of leverage. Hence, an

explanation of the obliquity of the axis of the neck of the astragalus and of the apparently retained mild inward slant of the calcaneal facets is clearly disclosed. The subsequent convergence of the two pressure streams upon the line of leverage from either side is a secondary essential phenomenon, in order that the lateral movement of each stream be neutralized and altered to a directly forward one in the plane of leverage action.

The proportionate strength of the inner to the outer streams of pressure is primarily controlled by the slope of the facets which support the astragalus. If the inward slant is increased, a greater amount of pressure is thrown upon the inner border of the foot and less upon the outer, thus losing a normal balance; while if the slant is decreased, the balance is lost toward the outer side. Were the facets level in so far as the lateral slant is concerned, then the Cuboid stream on the outer side of the foot would not be effectually counterbalanced by the Scaphoid stream, and when the latter became influenced by the lateral pressure of the flexor digitorum and posterior tibial muscles, it also would be directed toward the outer border of the foot. Such a condition would mean an inevitable distortion of the line of leverage, with a resulting hypertrophy of one or more of the outer metatarsal bones.

Consequently, the term "balance" as applied to the foot structure does not refer to muscle activity, but to the arrangement of the bones and ligaments which furnishes a stable base upon which body-weight can be supported with the least demand for muscular exertion, and propelled forward evenly balanced upon the lever axis. Since a distribution of pressure to the outer side of the foot is inevitable because the astragalus is superimposed upon the os calcis, the inward divergence of the Scaphoid stream must be regarded as the *primary* counterbalancing factor; and since that divergence is conditioned by the inward slant of the calcaneal facets, it therefore becomes obvious that the position of the os calcis is a most important element in "structural balance."

"*Diminishing Load.*" During locomotion there are two phases of decrease in body-weight upon the forepart of the foot, both of which may be included under this heading; they are (1) conversion of body pressure into momentum by forceful leverage action of the foot, and (2) assumption of weight by the opposite foot when during slow motion, as in walking, both feet are in contact with the ground at the same time.

The first-mentioned phenomenon is interesting in that the changing position of the foot during the course of a forceful step affords an increasing advantage to the muscles in their propulsive effort.

In the diagram, Fig. 5, the angle ABC represents the arched foot from the heel to the toes. A perpendicular line from B to the base line AC indicates that the advantage of leverage power is relatively 4 to 3 over weight; that is, if the weight at B is 120 pounds, 90 pounds of lifting power at A is sufficient to equalize the weight.

If to the 90 pounds of pull 10 more be added for active leverage, the resulting ratio would be 400 to 360, or 10 to 9.

In the second position A and B have advanced to A′ and B′; then dropping perpendiculars, a′C is approximately $\frac{7}{8}$ of AC, and b′C $\frac{2}{3}$ of bC. The resulting ratio becomes 350 to 240, or 10 to 7.

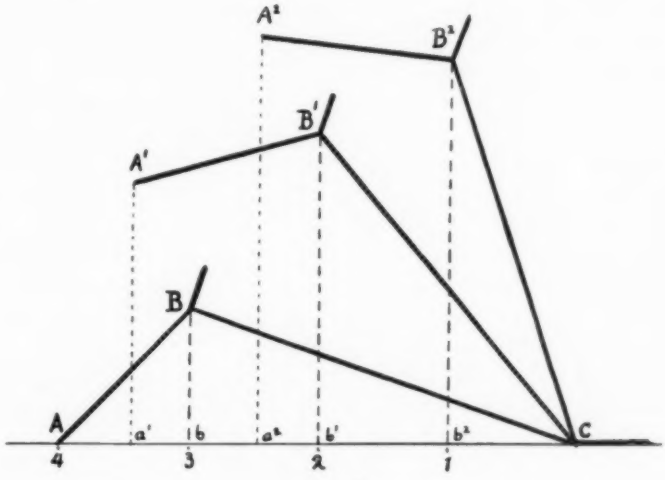

FIG. 5.

In the third position a²C is about $\frac{5}{8}$ of AC and b²C $\frac{1}{3}$ of bC. The ratio then becomes 250 to 120, or approximately 10 to 5.

In this rapidly decreasing ratio from 10 : 9 to 10 : 5 of the resistance of gravity upon the muscles, their force is correspondingly increased in propulsive power, while simultaneously, by the conversion of weight into momentum, the burden upon the foot is becoming diminished.

The second phenomenon which is characteristic of slow locomotion is very apparent in flat-foot patients. It consists, in its most extreme form, of a mere shift of body-weight from one foot to the other, the fore-part of the foot being scarcely used for leverage action. In the slow walk of a normal person, the shift is largely used, but with some active leverage.

As the gait becomes more rapid, the proportion of lessening load due to the shift and that due to increased momentum, is altered accordingly. In running, when only one foot is on the ground at a time, "diminishing load" is entirely the result of active leverage.

Muscle Action. When we estimate the relative strength of the forces that ordinarily involve the foot by the totality of pounds per minute, body-weight proves to be the greatest, and the force from the calf muscles is second. The bowstring action of the other muscles is "controlling" rather than "propulsive." We may make an exception of the long flexor hallucis in this classification, for it clearly combines both functions. The course of its tendon closely parallels the line of leverage so that its action is clearly propulsive and is continued into the important "toe snap." Its position beneath the sustentaculum tali causes it to be a prominent factor in helping to regulate the slant of the calcaneal facets.

The other four posterior leg muscles, the flexor digitorum, posterior tibial, and two peroneals, exert their major propulsive effort upon body-weight before it enters the foot by means of their positions behind the malleoli. Within the foot their function resolves itself chiefly into reverting the divided pressure streams upon the principal line of leverage. Their work is so divided and apportioned that each one is necessary to the proper action of the foot, and their insertions so scattered that, through their activity, there is a more complete coaptation of every segment to make the foot an efficiently functioning unit.

"Muscle Balance." The following simile gives a very clear differentiation between "structural balance" and "muscle balance" as the two terms apply to the foot.

A heavy bar is held vertically with one end resting upon the ground. In this position the bar is in balance and all of its weight is transmitted directly to the ground. Application of very slight pressure to the sides is sufficient to prevent the bar from losing its equilibrium; the lateral pressure, however, must be equal on opposite sides, otherwise the bar will be thrown out of balance. Thus, the bar may represent "structural balance," and the *equality* of the mild lateral pressure necessary to maintain its equilibrium represents "muscle balance."

Unlike the case of the bar, however, the controlling muscular action upon the foot presents other factors for consideration. The bar was a straight symmetrical object, while the foot and leg present a more complicated structure, and the strength of opposing muscle groups are invariably unequal. The factors which enter the consideration of muscle balance are probably most clearly shown in an analysis of antero-posterior sta-

bility. Here the relatively weak anterior group of leg muscles opposes the powerful ones of the calf; stability is maintained by these muscles acting as "stay ropes," and not by applied lateral pressure.

In erect stance, a perpendicular dropped from the center of gravity of the body does not coincide with a vertical plane between the centers of the ankle joints but falls anteriorly to this plane at a point between the two scaphoid bones; in other words, body-weight is not perfectly balanced antero-posteriorly upon the center of supporting contact, but is displaced forward (out of balance) and that displacement is held constant by tension upon the strong calf muscles. This pull, however, is a very slight one because the angle of "unbalance" is small.

The effect of this posture throws all the strain upon the calf muscles and relieves the anterior group from all stabilizing tension; incidentally the body is afforded the advantage of being maintained in equilibrium over the mid-point of foot length as a base for stability.

What constitutes "muscle balance" between these two groups?

A person standing erect can readily move his knees backward and forward (from hyper-extension to slight flexion), either alternately or together, with no appreciable effort so long as there is no lowering of the center of gravity of the body permitted. This action shows how slight is the strain of body-weight upon the muscles; and yet that slight strain is requisite, for it would be impossible to balance a person with paralysed legs upon his feet.

Again, in spite of the comparative weakness of the anterior group of muscles, their strength is nevertheless sufficient and perfectly adapted to maintain the normal relationship of the structures; a weakening of the anterior group, as from paralysis, would lead to an equinus deformity and secondary changes throughout the foot. (In connection with this latter point, it is interesting to note the range of position of the foot permitted by these opposing muscle groups when in a state of relaxation—when aided by gravity it amounts to fully one-half of the extreme arc of dorso-plantar flexion.) Such being the case, why is it that the stronger group does not continually encroach upon the weaker, ultimately to impair their normal relationship? The answer to this question seems to be found in an estimate of what constitutes "muscle balance."

The phenomenon appears to be dependent upon three major factors:

1. The proportionate strength of the opposing groups is conditioned by the burden habitually imposed upon them, and also by the relative degree of mechanical advantage or disadvantage under which they work.

2. A properly adjusted "moment of responsiveness" to stimulation from spinal centers, whereby the opposing action of the two unequal groups is restricted to purposeful and co-ordinated efforts.

3. The habitual exercise of normal function in order to preserve a proper ratio of functional demands and unaltered structural relationship.

The proportionate strength of opposing muscle groups as conditioned by the imposed burden and relative mechanical advantage is well demonstrated by the anterior and posterior muscles of the leg. That the posterior group performs the greater amount of work and must therefore be of greater strength is obvious; the shortness of the heel, however, compared to the length of the forepart of the foot, gives to the anterior group a decided mechanical advantage, because the insertions of the latter are more distant from the ankle joint, which compensates for the inequality in strength.

The "moment of responsiveness" refers to the period between the inception of a required muscle effort and its efficient performance, as distinguished from mere reaction to nerve stimulation. It is so evenly and perfectly maintained in the two groups that very slight muscular activity is required for the ordinary security of body equilibrium; the mild displacement of the center of gravity of the body which attends slight changes in posture or movements of the arms, etc., is instantaneously and effectually met by muscle response so faint that we are quite unconscious of it. The "moment of responsiveness" in either group may become sluggish, as by exhaustion, lost as by paralysis, or quickened by a state of over-stimulation, as in spastic conditions. A decided advantage is afforded to the more quickly acting group, which fact must be accounted for in "muscle balance."

Habitual exercise of normal function is necessary to the maintenance of the normal relationship in the opposing groups. If a proper ratio between muscle strength and the imposed burden is not kept constant, the stronger groups tend to shorten and encroach upon the weaker. This type of disturbance is seen in the equinus deformity of bedridden patients.

Although there may seem to be a very considerable range in the degree of importance of these three factors, disturbance in any one of them definitely impairs the phenomenon of normal muscle balance.

The lateral stability of body equilibrium is somewhat different from antero-posterior stability in the manner of its accomplishment. In the latter instance the legs (as viewed from the side) act as a single column and the effect of the greater calf muscles as "stay ropes" is required to hold the body in balance. Lateral stability of the body, however, is furnished by

two supporting columns (the legs), and the term "balance" is chiefly applied to the relative position of the foot to the leg.

The center of body-weight as it enters the foot is well buttressed by the sloping outer portion of the foot, but lies quite close to the inner line of structural support. This arrangement definitely affords a mechanical advantage to the weaker peroneal group, while the more hazardous nearness of the weight center to the arched inner border is guarded by a much stronger group of muscles. The normal arrangement of bone and plantar ligaments furnishes a staple structure upon which body-weight is actually balanced; consequently, as shown in the early part of this discussion, little functional demand is normally put upon the muscles as long as that balance is maintained.

The relative position of the foot to the leg when weight is assumed, is determined by the sub-astragaloid joint when the plantar ligaments (particularly the calcaneo-scaphoid) have become taut, and also by muscle balance. The lateral ligaments of the foot and ankle have little influence upon the ordinary working posture of the foot because they offer no resistance to free motion in the middle section of the arc of joint movement.

The action of the long flexor of the hallux during locomotion has already been given. The distinctive individual action of the other four posterior lateral muscles may be briefly described as follows:

The flexor longus digitorum helps to regulate the position of the plane of the calcaneal facets by upward pressure against the calcaneo-scaphoid socket, and arrests the inward movement of the Scaphoid stream by lateral pressure against that joint. The tibialis posticus sustains the inner stream of pressure and reverts it outward.

The peroneus brevis acts as a lifting force to lever the Cuboid stream inward and forward by means of the fifth metatarsal bone. The peroneus longus has a bowstring action against the outer stream to throw it inward, which is brought more prominently into play during forced locomotion when the stream principally follows the more direct channel through the cuneiform bones.

The intrinsic muscles of the foot need not be dealt upon at length as their function is less conspicuous and largely complementary. They furnish much of the resiliency and springiness of the foot. In the modern rigidly arched structure of man, ligamentous structure is the predominating element and the muscles are but slightly developed when compared with the intrinsic musculature of anthropoid feet; in fact, some of these muscles in the human foot are apparently undergoing an actual fibrous degeneration.

The anterior muscles of the leg and foot may also be passed over briefly as their function lies chiefly in "recovery" of the foot and adjusting its position for the succeeding step.

The Digits. Up to this time the toes have not been brought into consideration because they take no part in actual leverage function except as "anchoring" points for the tendons of the long flexor muscles. They are important factors, however, in both stance and locomotion.

In the former function the toes increase considerably the length of the foot as a base for stability, and in locomotion they give smoothness and grace to the slower movements, and a definite added impetus to rapid and forceful movements. In running and jumping the toe action may be likened to the "wrist-snap" that the good golfer has, and the others would like to acquire.

The effective snap of the toes is principally limited to the first and second, because the load is finally concentrated upon the two inner metatarsals. That load, however, it should be remembered, is not the full pressure of body-weight, because body pressure has been continuously decreasing by the conversion of weight into momentum.

The Transverse Arches. The only transverse section of the foot which deserves the name of an arch is located in the anterior tarsal bones near and including the bases of the metatarsals. In this region, cross section of the bones connecting the two longitudinal arches forms a rigidly arched structure. This condition obtains throughout primate feet and is observed in other mammals. It forms a very effectual vault for the protection of the most important blood vessels and nerves of the foot. In man this arch has become deepened by a downward bend of the inner section and a general narrowing of the foot which was associated with the development of the longitudinal arch.

The anterior or metatarsal arch is no more than a postural contour of the anterior portion of the foot and which has apparently been acquired as a result of the deepening of the tarsal arch. It is maintained by the subsequent contraction and tonicity of the transverse plantar soft structures of this part. The relative position of the metatarsal heads is changed and the arc becomes flattened under body pressure, especially when the foot assumes a position of pronation under weight.

EVOLUTION OF THE HUMAN MECHANISM.

The progress of development of the human mechanism can be very clearly followed in our interpretations of the gorilla foot as representing

a very ancient terrestrial period, and in the Neanderthal foot as an early human stage.

The Gorilla Foot. The mechanism of leverage in the gorilla foot has been discussed in the previous paper, but without any particular analysis of weight distribution (Fig. 6). In this type of foot, leverage action is imperfect and incomplete; body-weight is not carried entirely forward upon the metatarsal heads because the ligamentous structures have not

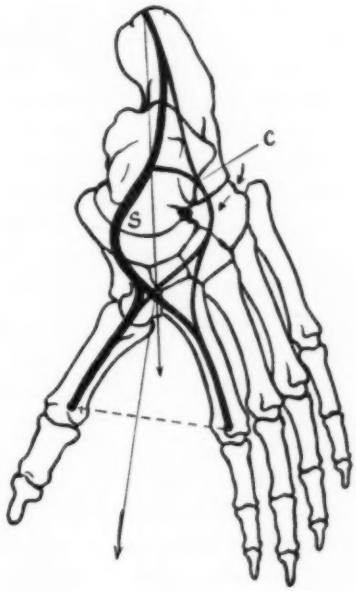

FIG. 6.

Movements of force in the gorilla foot. S, Scaphoid stream. C, Cuboid stream. Note the relative lack of function of the outer portion of the foot and the break in the line of leverage action.

been sufficiently developed, nor the tarso-metatarsal joints effectively adapted to withstand the inevitable forcible and painful hyper-extension of the metatarsal bones that would result owing to the animal's great bulk. Leverage action is definitely begun upon the metatarsal heads as the fulcrum, as is demonstrated by the strong development of its first and second metatarsals, but a portion of the weight is simultaneously shifted to the

opposite foot in the manner that we see in the gait of patients suffering from "flat feet."

Although the sloping position of the astragalus in the gorilla foot causes the greater amount of body pressure (S) to be deflected into the head of that bone, a certain portion (C) is transmitted to the outer side of the foot through the posterior facet and anterior process of the calcaneum into the cuboid. Here it is carried forward and inward toward the base of the two inner metatarsals principally by the action of the peroneus longus muscle.

The Scaphoid stream of pressure as the major one, moves forward through the scaphoid and inner cuneiform bone where it is joined by the Cuboid stream. Both streams divide to be transmitted to the ground by the first and second metatarsal bones. This final movement is not directly forward in a direction parallel to the inner border of the foot as in man, but obliquely inward as described in the previous paper.

The Neanderthal Foot. The foot of prehistoric Neanderthal man shows differences that are clearly intermediate between the modern human and the gorilla characteristics. Such intermediate resemblances are also to be observed throughout the entire Neanderthal skeleton. This ancient race inhabited Europe between 25,000 and 75,000 years ago. Although several almost complete skeletons of these individuals have been discovered, none of the actual material has ever been brought to this country. Prof. J. H. McGregor, of Columbia University, has kindly presented me with casts of the left astragalus and os calcis of one of these prehistoric human beings, and allowed me the privilege of studying the casts of four other Neanderthal astragali. The common points of difference from the modern type have been analyzed to learn the indicated variation in foot function. Fortunately the casts are of the two bones which are of the greatest value in a determination of the mechanics of the foot and their peculiar characteristics give a very clear conception of the more primitive distribution of pressure through the anterior part of the early human foot.

A brief outline of the analysis may be given as follows: The inward placement of the head of the astragalus to the body and the marked inward slope of the facet on the sustentaculum tali of the os calcis definitely shows that a very strong stream of pressure was thrown to the inner border of the foot. This would indicate that under body-weight the first metatarsal bone would be distinctly divergent, and with its divergence the second metatarsal would receive a greater share of body-weight than in the modern type of foot. There would consequently be less function

performed by the outer side of the foot. The Neanderthal foot was undoubtedly a broad, low-arched one with a relatively shallow and flaring transverse tarsal arch; the Scaphoid pressure stream was stronger and the Cuboid stream weaker than in the modern foot; the outer border of the foot definitely entered the leverage action but not to the same degree as at present (this would naturally be associated with a less stoutly developed fifth metatarsal bone); the final separation of the reunited streams to follow the diverging first and second metatarsal bones would be associated with a retention of the primitively acquired stoutness of the second metatarsal.

Such an interpretation assigns to the Neanderthal foot a stage of development in its functional mechanics that was clearly intermediate to that of the modern human and of the very early prehuman terrestrial stage, as indicated by the characteristics of the gorilla foot.

Variations in the Human Type of Foot. The concensus of opinion among scientists allots to all the living races of man (Homo sapiens) a single point of origin; hence, a common starting point in foot development. All mankind are terrestrial bipeds; therefore the mechanics of human foot function is fundamentally identical in all individuals. Just as man presents differences, however, in stature and facial characteristics, in a like manner his feet have had the opportunity to develop hereditary or acquired differences of minor character that yield a wide range of variation.

But function has not worked alone in evolving the human foot, for it has ever been associated with other factors. Certain combinations of these factors give rise to superficial modifications which to some measure depart from the effects of a different group of factors. For example, the passing of ages over a segregated group of individuals gradually promotes the development of distinctive racial characteristics; customs and habits, such as shoe-wearing and non shoe-wearing, and sedentary or active modes of living, have a direct influence upon foot development, as also does life in the open or in congested centers of population; in fact, so numerous are the factors, congenital and acquired, mechanical and physical, racial and individual, environmental and internal, and their various combinations, that it is not surprising that every foot should have its own distinguishing characteristics. There are high, medium, and low arches; feet range from slender to broad in proportion to their length; some are very flexible, others rigid; there are variations as to the respective lengths of the toes and in the relative position of the heads of the metatarsal bones, or in the relative stoutness of the separate shafts. Any part of one foot structure may show elements of difference from other feet.

When these differences are analyzed, however, they are seen to be only superficial modifications as long as there exists no appreciable disarrangement of the functional mechanics of the foot, but when the latter occurs, modifications become more than a mere variation, for they then tend to follow a steady course toward an actual deformity characteristic of the nature of the fault.

From a clinical aspect, the variations in human feet may be classified in two main groups: (1) Normal variations, which consist of superficial differences that do not affect the foot's normal mechanism; and (2) abnormal variations, which are no less than secondary deformities resulting from some disturbance or disarrangement of the normal distribution of forces within the foot structure.

MECHANISM OF THE NORMAL FOOT AND OF FLAT FOOT.

PART II.

Disorders of the Foot. Practically all disorders of the foot involve in some way its mechanical function. The disturbance in the distribution of body-weight may consist of a temporary "favoring" of the foot in order to avoid or to lessen irritation, as from a sensitive blister or corn; it may be the improper distribution of body-weight associated with an habitual use of faulty posture; or it may result from a disorder of some of its functional parts, as in paralysis or fracture. Where the disturbance is purely of a temporary or superficial character, normal function returns as soon as the source of irritation is removed. But, in disorders of a more permament and deeper nature, progress is seldom spontaneously corrective but tends toward an increase of the disorder.

The most important part in the examination of a foot is the final *visualization* of its internal functional phenomenon as indicated by the outwardly visible signs and symptoms—or, in other words, an intimate conception of just how the balance and function of the foot is impaired. Such an analysis furnishes the best working basis upon which to determine the mode of treatment that will insure the most favorable results.

The essential phases of functional disturbance in the foot are well presented in the analysis of its static disorders. Other causes of foot disorder merely effect a modification of the same faulty mechanics.

The vertical plane of leverage action upon which the forces acting upon the foot are normally balanced, may be considered the dividing line

by which the majority of functional disturbances may be separated into two large groups: (a) disorders in which an increased burden is thrown to the inner side of that line, as in flat foot and its allied disorders, and (b) those conditions in which there is an overbalance of forces to the outer side of the foot, as in club feet and other varoid disturbances.

Of the two groups it is quite natural that the former should be the more common because the inner arched border does not present the secure, laterally buttressing structure which the sloping outer longitudinal arch affords to the foot.

Flat Foot. The term "flat foot" is here used to include the intermediate stages of pronation through which the foot passes from its normal balanced position to the final stage of rigid and characteristic deformity. The ultimate alterations of the bones clearly register the influence of the faulty stresses and disordered movement of forces within the foot which are produced by the faulty posture of the earlier stages.

Pronation, as an inward rotation of the foot under body-weight, is a movement that occurs upon the heel and head of the first metatarsal bone as the axis of rotation. This movement of the foot extends upward to the astragalus, but does not directly involve that bone; that is, the astragalus, being firmly held between the two malleoli, cannot be included in the rotation of the foot, but follows an inward swing with a depression of the head. Viewed from above (Fig. 7), the vertical plane of the inner border of the foot changes from practically a straight line ABC to a foreshortened triangle AB′C. The short posterior side of the triangle represents the new position of the longitudinal axis of the calcaneum; the associated inward swing of the axis of the astragalus carries the leg bones with it. The inward rotation of the os calcis (a) increases the inward slant of its superior facets, so that they act as lubricated inclined planes to deflect body-weight off the side of the foot; (b) causes the lowest point in the scaphoid socket toward which the head of the astragalus gravitates to move rapidly inward with each degree of pronation, and (c) carries the center of body pressure in the astragalus toward the inner border of the foot and away from the outer.

It is therefore seen that in pronation all the initial and primary factors which create the disturbance of the normal function of the foot are intimately associated with the altered position of the os calcis. The pronated posture increases the Scaphoid stream of pressure, as described in the previous paper, and diminishes correspondingly the Cuboid stream; the resulting effects upon the function of the fore part of the foot are clearly secondary to the disturbance that originates posteriorly to the mid-tarsal joint.

Regarding the quadrant of a circle as the arc of pronation, it will be noted that the early movement is essentially a lateral one, and that only after the center of the quadrant (45°) is passed does downward movement predominate. Hence, in the early stages of disorder the normal position of body-weight to the supporting plantar ligaments is generally maintained. As the movement continues, however, body-weight is slid off of the *direct* support of the powerful plantar ligaments and upon the weaker lateral ligaments and muscles.

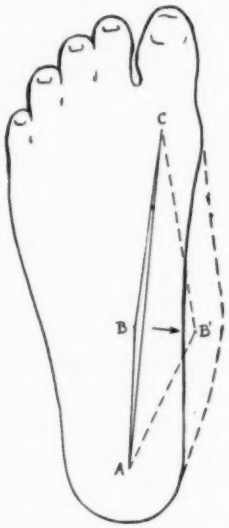

FIG. 7.
Normal (ABC) and pronated (AB'C) positions of the inner line of support.

Functional Relationship of the Supinating Muscles and Plantar Ligaments. We are here led to a consideration of the parts played by the soft structures in the actual support of body-weight. There seems to be a very noticeable degree of confusion as to the functional relationship of the plantar ligaments and of the supinating muscles of the foot in the support of body-weight. The muscles are apparently given far more credit than they deserve. The statement that the flexor and posterior tibial muscles are not weight-supporting structures may seem exaggerated, but it is a true statement of facts. Body-weight is sustained and carried

by the plantar ligaments, while, under normal conditions, the action of the muscles is merely *controlling*.

The following observations bear out this statement:

1. The total pulling power of the flexors and posterior tibial muscle is but a fraction of body-weight, and under sustained pull their power rapidly dwindles to a very insignificant amount.

2. No one has ever attempted stretching (under anasthesia) of the plantar ligaments of even a small child's foot without being forcibly impressed with the great strength of those tissues.

3. The foot of an adult cadaver (of about 150 pounds) was subjected to a direct downward pressure of over 300 pounds. With the application of the first 150 pounds the arch of the foot assumed the normal contour as under body-weight; as the pressure was raised to 300 pounds it was attended with only a mildly increased sinking of the arch with no apparent injury to the tissues. The foot regained its normal contour immediately when relieved of the pressure.

The foregoing observations are sufficient to demonstrate that the plantar ligaments are sufficient in themselves to sustain body-weight, and that the muscles, at best, could afford but little assistance in protracted strain even if their help were required. Moreover, in regard to the regulation of the position of the center of body pressure upon the foot, the same muscles have little more than an auxiliary rôle. A shift of body-weight to the outside of the foot is accomplished largely by the strong biceps through an outward rotation of the leg bones which carries the astragalus with it. The flexors and tibialis posticus aid in this action by a simultaneous elevation of the inner border of the foot. That they are incapable of accomplishing this action alone is clearly demonstrated in the following tests:

1. When a person stands erect with the feet parallel, the shift of weight to the outer side of the foot seems to be accomplished by the action of the leg muscles against the inner side of the foot. Let him now rotate the legs outwardly so that the feet are practically on a straight line with the heels close together. This position will throw out the action of the biceps but does not alter the relative position of the legs to the feet to interfere with the action of the leg muscles. It will then be found impossible to shift body-weight to the outer border of the foot by the single action of the latter muscles.

2. A second test is to assume a semi-squatting posture with the feet again placed parallel and heels firmly upon the floor. In this position the acute flexion of the knees will materially reduce the action of the biceps.

The outward shift of body-weight will be found extremely difficult, which is not the case as soon as the erect posture is again resumed.

Although these observations clearly contradict any claim of the leg muscles as weight-bearing structures, they do not infer that those muscles are not of utmost value in the normal function of the foot. In fact, the comparative weakness of the muscle element is most useful in emphasizing how definitely the phenomenon of *balance* applies in the physiology of the human foot. The exact functional relationship of the muscles and ligaments is quite clearly shown in the accompanying diagrams.

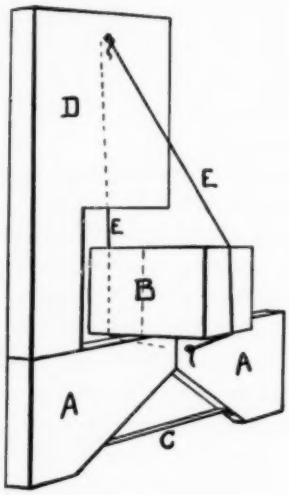

FIG. 8.

In the first diagram, Fig. 8, the vertical axial plane of support (coincident with the plane of leverage action) is represented by two irregularly shaped plates AA which are cut away beneath the block B (representing body-weight) to form an arch. The arched formation is maintained by the tension band C which is fastened to both plates so as to prevent them from separating. The tension band represents the plantar ligaments of the foot, and upon it all the strain of the weight is referred.

The upward extension D represents the leg bones, and from it stay ropes EE are dropped around each side of the block to be fastened to the supporting structure beneath. No real strain is placed upon the stay

ropes, which may be compared to the lateral muscles of the leg, because the weight B rests in balance.

In the next diagram (Fig. 9) the block has been shifted slightly to one side. It is no longer in perfect balance. The smaller overhanging portion of the block counterbalances a similar portion on the other side as indicated by the broken line; the small section f which extends beyond that line comprises the load which is now thrown upon the stay rope. The weight of this latter portion, however, as shown in the third sketch, is transmitted by the rope to the upper extension D and moves through

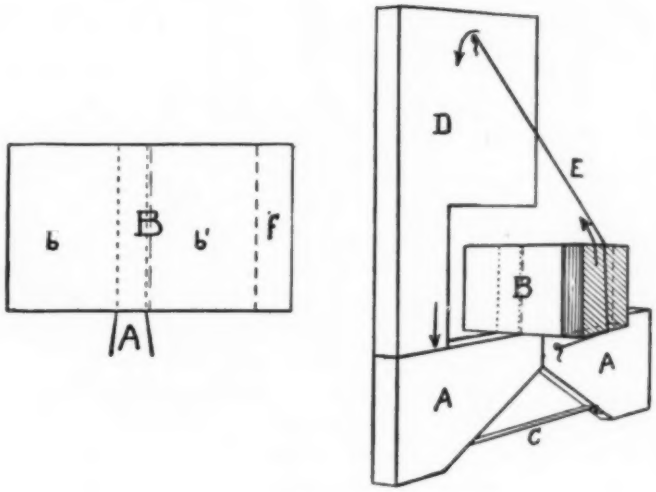

FIG. 9.

the latter back upon the supporting structure A. Consequently, no lessening of the burden upon the tension band C takes place.

In a general way these diagrams illustrate the balance and shifting of body-weight as evidenced by the normal foot. The burden is usually held in balance and any incidental disturbance resulting from movements of the body is so transient and mild that it does not subject the lateral structures to unduly prolonged or severe strains.

Disordered Function. The diagrams of Fig. 10 show the derangement which results from the faulty posture of pronation.

The supporting structure A is now placed obliquely while the extension D is retained in its upright position. The block B is forced to assume a sloping position because of the slant of the surface which supports it. It may be assumed, also, that the block has been shifted laterally to some extent.

The diagram of the side of the block demonstrates the altered distribution of weight. A represents the slanting upper surface of the supporting structure; a, the portion of the block which receives direct support from A; b, the overhanging portion of the lighter side; b′, the corresponding

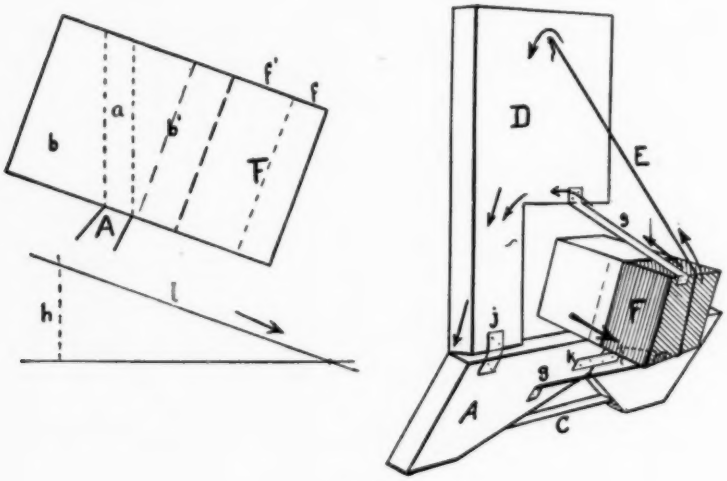

FIG. 10.

counterbalanced portion. The large remaining section F consists of two parts: f, which was described above as due to the lateral shift of the block, and f′, which is the result of the inward displacement of the center of gravity of the block owing to the obliquity of the supporting structure. A very considerable increase in the burden upon the lateral structure is observed in the size of the total section F.

There is still one other factor to be included in the calculation—the effect of the "frictionless" inclined plane on which the block is supposedly supported. The amount of resistance necessary to prevent the block from sliding to the side is determined by the height of the slope h in its ratio to the length l. If a mass weighing 120 pounds rests upon a friction-

less plane whose slope $\frac{b}{l}$ is $\frac{1}{3}$, the resistance necessary to prevent movement of the mass would be $\frac{1}{3}$ of its weight, or 40 pounds. In this manner we can obtain some idea of the inward thrust of body-weight to which the lateral structures are also subjected as a result of pronation.

In the larger diagram, the phenomena just described are applied. The short strips g, j, k have been added to represent lateral ligaments of the ankle and foot. Upon the stay rope E and the strips gg now falls a greatly augmented burden, which consists of the load F combined with the lateral thrust. The total burden is transmitted to the superstructure D and then to the joint where it is finally reverted upon the supporting structure. At this joint a breaking strain is exerted upon the strip j, as the weight coming from above tends to tear open the joint by causing a collapse of the sloping structure A. Longitudinal strain is also put upon the strip k because of the tendency of the two halves of the support A to separate laterally beneath the superimposed weight.

"False Load." It has probably been noted that in the foregoing analysis an attempt has been made to clearly differentiate between the burden of body-weight which is constantly borne by the plantar ligaments and the abnormal burden that is thrown upon the lateral structures as a result of faulty posture. The latter burden can be very suitably termed a "false load," as it is entirely distinct from and independent of the natural burden borne by the foot. "False load" is a new, abnormal development that may be likened to a neoplastic growth—an invisible functional tumor, both useless and vicious!

There is no compensatory element that might justify its existence, for the "false load" thrown upon the lateral muscles and ligaments in no way relieves or lessens the burden upon other structures; to the contrary, it is entirely a destructive factor by which the tissues of the side of the foot and ankle are subjected to increasingly harmful stresses of body-weight which they are incapable of withstanding; they yield to the strain and in yielding they magnify the strain which is breaking them down.

It might be a proper simile to compare the condition with the case of a camper who carries twenty pounds of equipment comfortably upon his back and shoulders. The burden does not greatly affect his strength nor impede his movements. But let him attempt to carry the same weight in his extended arms and he will quickly become exhausted. In both instances the total weight upon the camper's feet is the same, but in the latter case the burden is carried disadvantageously and the resulting strain is entirely distinct from and independent of the support furnished by his legs.

The Fore Part of the Foot. So far the discussion has largely been centered upon the posterior half of the foot. In that part the disordering of function primarily takes place and the disturbance is carried forward as a secondary effect. Pronation causes an increase of the Scaphoid pres-

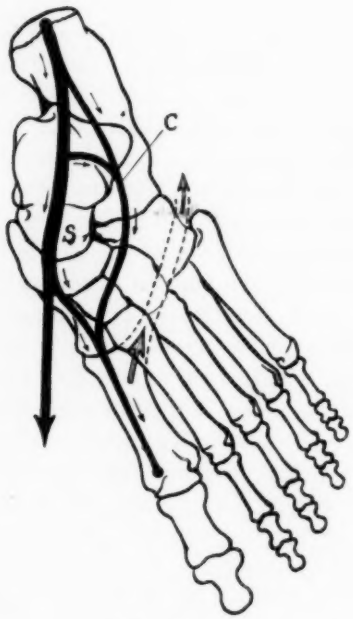

FIG. 11.

Movements of force in a pronated foot. Note the heavy Scaphoid stream (S) which continues over the side of the foot, and the small Cuboid stream (C). The outer side of the foot is comparatively functionless and all of its pressure (C) follows the Cuneiform route, practically none going to the metatarsal bases. The strong pull of the peroneus longus muscle is indicated by the shaded arrows.

sure stream and correspondingly diminishes the Cuboid stream. Body-weight which under normal conditions is distributed to the outer side of the foot tends to become concentrated upon the inner longitudinal arch. The vertical plane of support upon which body-weight was formerly balanced, and which practically coincided with the vertical plane of lever-

age action (located between the first and second metatarsal bones), is now moved inwardly to a line from the heel through the inner side of the head of the first metatarsal. The plane of support is now oblique instead of vertical.

In the faulty distribution of body pressure of a pronated foot in stance, practically all the weight of the body is borne by the heel and first metatarsal bone. The outer border of the foot tends to be rolled upward from

FIG. 12

X-ray of normal (left) and markedly flattened (right) feet. The lessened density of bone of the outer side of the flat foot is very noticeable when compared with that of the normal foot. Compare the denser area of the flat foot with the movements of force as shown in Fig. 11.

firm contact with the ground and consequently placed in an impossible position for the transmission of body pressure to the ground.

In locomotion (Fig. 11), the faulty strains become more apparent. Propulsive force from the heel acts against the inwardly displaced weight center in the astragalus to project body-weight off the inner side of the foot; the inward slant of the calcaneal facets adds strongly to this inward thrust. This oblique movement is further promoted by a change in leverage action.

Formerly leverage action moved directly over the fulcrum—the heads of the first and second metatarsal bones; now, however, body-weight is carried upward and obliquely to the inner side of the fulcrum. This seems to clearly explain the tendency of weak feet to assume an out-toeing posture. Momentum is now created obliquely from the side of the foot instead of over its long axis, and the portion of body-weight which is transmitted to the fulcrum is carried almost entirely by the first metatarsal. The smaller Cuboid stream of body pressure scarcely extends to the outer metatarsal bone, but proceeds through the cuboid and cuneiform bones almost directly toward the base of the first metatarsal. Evidence of this is very clearly shown by the difference in the density of the tarsal areas in X-ray study of the skeleton of a normal and of a flat foot (Fig. 12).

Deformity of the Bones. Disarrangement of the normal movements of forces through the foot structure ultimately leads to distortion of the bones, as joint surfaces are thrown into uneven contact with the disordered thrust and pull of muscles and connecting ligaments.

The alterations are particularly notable in the midtarsal area (Fig. 13). The most conspicuous deformity consists of an outward "break" of the foot at the tarso-metatarsal juncture which allows an actual downward sinking of the arch in the final stages of flat foot, instead of a continued and more extreme pronation. This outward "break" is apparently the result of the obliquely outward pull of the tibialis anticus and peroneus longus muscles, both of which have their insertions at the base of the first metatarsal. Their pull is directly opposite to the direction of the obliquely inward thrust of body-weight in the scaphoid. The cuneiform bones are located between these oppositely moving forces. The effect is very distinctly shown in the lozenge-shaped middle cuneiform. In the normal foot the longitudinal axes of the three inner metatarsal and their respective cuneiform bones, and of the two outer metatarsal and the cuboid bone, are practically continuous as straight lines. In the flattened foot, however, the break in the line of their axes is very apparent.

Changes of greater or lesser degree may be observed throughout the bones of the foot. They involve the astragalus in a distortion of its trochlear surfaces, an altered axis of the neck, and an increased outward lateral extension of the facet on the head. The scaphoid bone is forced downward so that its tip flares noticeably inward when the position of pronation is reduced. The sustentaculum tali becomes depressed in its relative position to the body of the os calcis. The long axes of the shafts of the metatarsal bones tend to become convex toward the inner side of the foot, and cross section of the shafts shows a considerable increase in the inward

oblique diameter as compared to the vertical measurement. The transverse metatarsal arch becomes definitely flattened.

When changes in the contour of the bones take place, disordered function becomes fixed; the movements of force have then become definitely altered by these changes, so that it is impossible to re-establish the original

FIG. 13

FIG. 13. Photographs of the same specimens, showing the bone changes of flatfoot deformity. Compare the break in the axes of the tarsal and metatarsal bones of the flat foot to their straight continuation in the normal foot. This break and the Cuneiform distortion is clearly explained by their position between the oppositely moving forces (the thrust of body-weight and the peroneal pull) as shown in Fig. 11.

lines of movement. The bone alterations are comparatively slow in starting, for a very appreciable amount of "false load" must be developed before they may occur. Acute inflammatory reaction of the soft tissues hastens these bone changes by lowering the resistance of the tissues to faulty stresses, and by a mild extension of the inflammatory process to

include the bones, thereby making them more susceptible to the effects of uneven contact.

It is unnecessary to dwell upon the bone changes in further detail. The matter of principal importance is recognition of the wide dissemination of these changes which, although not markedly prominent in any single bone, nevertheless, in their totality may produce a very severe deformity of the foot.

ACUTE FOOT STRAIN.

Earlier in this paper the arc of pronation has been referred to as a quadrant in which downward movement predominates only after the middle of the arc has been passed. This comparison must be modified somewhat when applied to the process of foot disorder (Fig. 14).

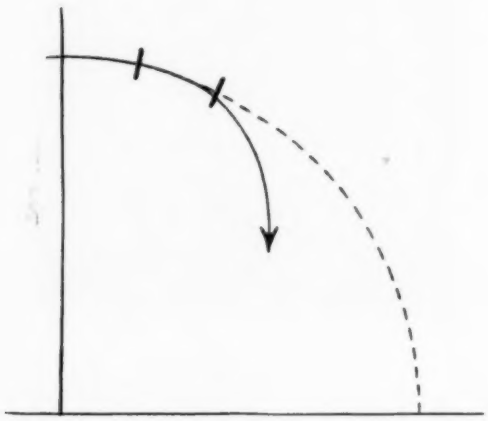

FIG. 14.

The almost completely horizontal motion that first takes place may be considered characteristic of the first stage of pronation.

During the second stage the continued lateral movement is associated with increased downward movement. At some point in this second portion of the arc, the structures tend to succumb to the influence of the abnormal burden thrown upon them, and subsequent movement breaks away from the original arc of pronation and follows a more directly downward sinking.

It is at this period and immediately following it that painful foot strain occurs, with stretching of the various lateral and interosseous ligaments, periosteal irritation, muscle exhaustion and spasm, and uneven contact of the bones. The entire body-weight tends to become concentrated upon the calcaneo-scaphoid ligament, against which the head of the astragalus is forced as a wedge. This period of acute strain may occur early in the second stage or late, and may be manifested suddenly or gradually, according to the nature of the exciting cause or incident.

Foot disorder is very properly likened to dental decay. The cavity in a tooth may have existed for a long time before it becomes sensitive—but the defect naturally began when the cavity started. In the same way disorder of the foot begins with the early habitual use of the faulty posture—acute strain is the painful climax of the vicious circle which has been gradually destroying the integrity of the foot.

The separation of "acute foot strain" from the disorder of Flat Foot is purely upon a clinical basis and due to the prominence of subjective symptoms; from the viewpoint of pathology, acute strain is merely a period in the progress of the advancing disorder.

SHORTENED CALF MUSCLES.

A shortening of the two major muscles of the calf, gastrocnemeus and soleus, with their common Achilles tendon, is usually associated with the changes leading to flat feet. The contracture may be secondary to the alterations in the feet, or it may be the primary cause of the foot's disorder.

Two of the most conspicuous etiologic factors of arch trouble are directly traceable to a primary shortening of these muscles; namely, the use of high-heel shoes and a long period of illness in bed during which the feet have been allowed to maintain a position of equinus or "toe drop."

An analysis of the effect of wearing high-heel shoes will best demonstrate the injurious action of the shortened muscles.

The normal range of dorso-plantar flexion is estimated to be about 75 degrees, 15 degrees dorsally and 60 degrees plantarly. (This movement is determined by the angle of the outer border of the foot to the leg with the sole slightly inverted and *with the knee fully extended.*)

The angle of elevation of the heel caused by a 2¼-inch shoe heel is 25 degrees—considerably more than the ordinary range of dorsiflexion (Fig. 9). Shortening of the calf muscles to accommodate the leg to the continuous use of high heels, therefore, tends to completely obliterate normal dorsiflexion. The actual shortening of the lines A′D′, representing the position

of the calf muscles with a high heel, is about one inch compared to the line AD, the normal position. The vicious effect of the muscle shortening upon the arch of the foot, however, is not during the use of high heels but when they are not used, as shown in the second diagram. Here the shoe heel is removed and the heel lowered to the ground. The right angle position of the foot now takes up all the slack in the calf muscles and heel cord. Any attempt to advance the leg bone, as is necessary in locomotion, must be attended with one of three conditions: (1) an immediate elevation of the heel, (2) a forced stretching of the calf muscles (which is practically impossible, owing to their great strength), or (3) a lowering of their sites of origin by a sinking of the leg bones. The effect of the shortening would be practically the same upon a man's foot if he were to fasten a two-inch block to the sole of each shoe, or were to attempt to walk barefooted with a one-inch block strapped beneath the ball of each foot.

In young athletic women with strong resisting tissues, the quick elevation of the heels is quite noticeable when heelless or very low heel shoes are worn. This manner of using the foot is only temporary, however, because eventually a lowering of the leg bones takes place to compensate for the shortening. It is accomplished by a downward thrust of the astragalus upon the shelf-like sustentaculum tali, forcing the os calcis to roll inward and downward from C to C′, and the resulting increased inward slant of its facets immediately gives rise to the disorders of pronation which have already been described.

In cases where primary shortening of the calf muscles has resulted from some other cause, the faulty mechanics is the same as that already described. A secondary shortening of these muscles may be looked upon as the opposite condition; in these cases, primary foot disorder permits a sinking of the origin of the calf muscles, producing a laxity which is subsequently taken up.

SUMMARY.

In the analysis of Flat Foot and its allied disorders, the following points stand out as the most important ones:

1. The disorder is essentially and primarily a faulty posture of the os calcis, by which an *improper distribution* of body-weight and muscle force is transmitted to the fore part of the foot through its two channels, the scaphoid and cuboid bones; in the fore part of the foot, functional derangement and subsequent deformity is purely secondary to the improper distribution of forces thrown upon it from the posterior half.

2. The nature of the disorder is a loss of the normal balance of the forces passing through the foot so that the unbalanced structure is caused to roll inward (pronate). This movement increasingly exaggerates the faulty distribution of pressure and at the same time breaks down the resistance of the tissues which attempt to oppose the abnormal movement. Hence a vicious circle is formed.

3. The plantar ligaments are the only real weight-supporting soft structures of the foot; the lateral muscles and ligaments are merely controlling in their actions and incapable of sustaining any considerable amount of body-weight.

4. The early movement of the faulty posture is almost directly lateral; the resulting progressive slant, however, of the calcaneal facets causes an inward shifting of body-weight and the creation of a strong lateral thrust, which together constitute an overpowering burden upon the relatively weak lateral structures.

5. This burden is termed a "false load" because it is a new and abnormal element of strain thrown upon lateral structures and by them is merely transmitted back again upon the leg and foot. This "false load" constitutes the pernicious element of this type of foot disorder and should be viewed as a vicious development that is comparable to a neoplastic growth; it has no useful purpose, and is solely destructive in its effects.

6. Bone changes ultimately occur which accord with the disordered movements of force and the uneven contact of joint surfaces. The changes are widely scattered to all the bones of the foot, each bone presenting a relatively small share of the total deformity.

REMARKS ON TREATMENT.

The full course of flat-foot disorder from the period of its inception may be separated into two distinct stages from a prognostic as well as a therapeutic standpoint:

1. An interval of *Functional Deformity* when the element of "false load" as a destructive agent predominates and tends to progressively break down the integrity of the foot by causing an increasing disorder of its normal mechanics.

2. A stage of *Structural Deformity* when distortion of the bones occurs and makes permanent their faulty relationship to the movements of force.

Since the disorder is a progressive one, there is no distinct point of demarcation, and other factors, such as the age and habits of the individual, influence the response of such border-line cases to treatment. The ideal

time to correct this type of disability is, naturally, prior to the beginning of bone change, but lack of knowledge on the part of the public makes early recognition and treatment a comparatively infrequent occurrence; the vast majority of patients who present themselves for professional care do so after structural alteration has become well started.

The early stage of functional deformity is completely correctable. The development of "false load" is the product of an ever-widening vicious circle. The principal factors which permit the enlargement of the circle are the increasing slant of the calcaneal facets (a loss of "structural balance") through a stretching of the ligaments of the inner side of the foot and ankle (especially the calcaneal scaphoid and deltoid), and a disturbance of "muscle balance." The latter is caused by a stretching and exhaustion of the supinating group with a resulting retardation of their "moment of responsiveness" which is more completely overbalanced as the condition becomes acutely painful, by a quickening of the peroneal responsiveness associated with the spasticity of the latter group.

Hence, there are two definite points of attack upon the disordering process: (1) by maintaining the os calcis in its normal position, which will prevent abnormal strain upon the lateral ligaments and muscles, permitting the stretched ligaments to shorten and protecting the muscles from a burden in excess of normal requirements, and also insure a proper distribution of pressure to the fore part of the foot; and (2) by actively promoting a return of normal muscle balance.

In cases where the condition has been allowed to develop a very acute subjective stage, a short period of complete rest of the tissues will greatly hasten the recovery of muscle control; it affords a complete suspension of all strains and allows perfect relaxation which acts to advantage in restoring the strength and normal responsiveness to the overworked and weakened supinators, and it also allays the spastic over-stimulation of the peroneals. This rest should be followed by special exercises for the feet, but not at first against body-weight.

One of the most important phases of treatment is the complete and constant inhibition of faulty lateral strains in so far as it is possible to do so. This is accomplished by maintaining the heel in its normal posture by one of the methods given below. An attempt to correct the disorder by restoration of muscle balance alone is to work against a big handicap, for such a method continues to impose upon the weakened muscles the double burden of abnormal posture. Better and quicker results are to be obtained by adapting the load to fit the strength of the muscles, or, in other words, by first securing "structural balance."

As shown in the foregoing studies, when body-weight is "in balance" (structural) very slight muscular effort is demanded to maintain that balance. This fact would seem to be the keynote of treatment—that is, first obtain a proper distribution of weight throughout the foot (which immediately eliminates "false load" and its pernicious effects) and then employ such therapeutic measures as will facilitate a return of the tissues to their normal condition.

Shoes are primarily a foot covering. Efforts have been made to commercialize them as corrective articles, and with great success financially, because of the wide prevalence of foot disorders, although with but questionable results from a therapeutic standpoint. There are two widely differing principles employed in the so-called "orthopædic" types of shoes: (1) an arch-supporting shoe which is not corrective in its principle but which may be palliative if it happens to conform properly to the contour of the arch, and (2) a very flexible type whose alleged corrective properties are assigned to the freedom of muscle action—a theory that coincides with an attempt to tame a wild horse by giving it a loose rein.

All newly-purchased shoes have a temporary corrective influence upon the foot by reason of the stiffness of the counter in an unused shoe, and especially if it fits the heel snugly. This property, however, is short-lived, for no pliable shoe-upper can resist the strains of body-weight very long in the presence of moisture from within or outside the shoe.

The selection of a shoe in the treatment of a case of foot disorder would, therefore, not be based upon its corrective properties, but is made upon its lack of qualities harmful to the foot.

Restoration of "structural balance" in the earlier stages of disorder is usually obtained by shoe alterations—wedging up the inner side of the heel and, if desired, "infloating" it. As in the *new* shoe, however, the effectiveness of the heel wedge is absolutely dependent upon a snugly fitting heel counter. If the counter is too wide or becomes spread, the heel (foot) slides against the outer portion of the counter and the ankle is permitted to roll inward. It must be remembered that the os calcis presents a rounded bearing surface, and, in addition, about a half-inch of pliable soft tissue separates the bone from the slanting heel seat; consequently, heel wedges act chiefly through the outward obliquity of closely fitting counters rather than by any influence the slant of the heel surface might tend to exert directly upon the rounded under surface of the bone. An inner wedge of the sole may also be used in more advanced cases but should be discontinued as soon as an improved posture of the foot is ac-

quired, because a better relationship of the anterior part of the foot to the heel is obtained when only the heel wedge is used.

The height of the shoe heel is of particular importance in the treatment of foot strain associated with shortened calf muscles, so common in women. A proper height of heel to prevent lateral strain can be easily determined by estimating the angle of dorsiflexion permitted by the ankle. In locomotion the leg should be allowed to move forward through an arc of 20

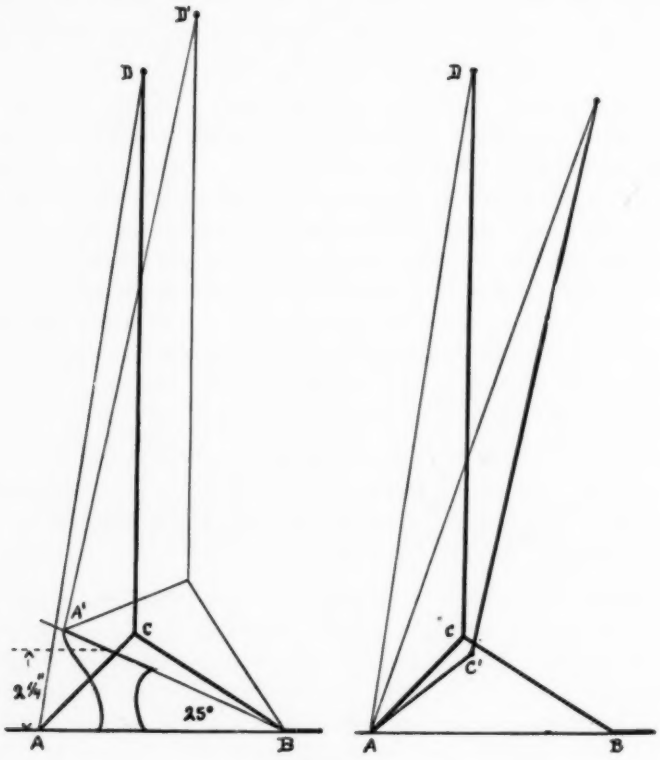

FIG. 15.

degrees from its perpendicular position, before it is stopped by the calf muscles. In a right-angle contracture this arc would mean a minimum two-inch heel; five degrees of possible dorsiflexion requires a one and one-half-inch heel; ten degrees (common in men) is most comfortable with a one-inch heel. (The angle of flexion is determined with the knee held straight and the sole slightly inverted.) The above ratio of heel height

to dorsiflexion has been found to constitute a very satisfactory working rule. A little care in measuring the angle of flexion and in prescribing the indicated height of heel accordingly will produce very gratifying results. Heelless shoes and slippers are strongly contra-indicated in this class of cases; pumps or "mules" should replace bedroom slippers.

A most effectual method of maintaining structural balance is the customary adhesive strapping. It is very positive in its action but, of course, cannot be used indefinitely because of its tendency to irritate the skin which causes objection from the patient. An arch pad of felt of suitable thickness may be included in this adhesive dressing.

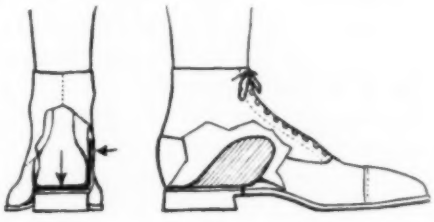

FIG. 16.

As a result of the preceding analysis of flat foot, in which the disorder is viewed as a primary inward rotation of the os calcis, and the strains as an abnormal inward thrust of body-weight in the area of the astragaloscaphoid joint, a somewhat different method of treatment has been devised and tried out very successfully. It employs the principle of a right-angle lever by which the body-weight is employed as an active force to automatically resist the faulty inward thrust and displacement of the foot. A piece of light sheet metal or other rigid material is bent to a right angle; one part is placed under the heel (within the shoe) and the other portion extends upward along the inner side of the foot toward the astragaloscaphoid joint. The angular bend acts as a fulcrum. Body-weight, first falling upon the heel piece, immediately stabilizes the vertical extension in its position so that any tendency toward a faulty inward movement of the foot is immediately resisted. In this manner the foot is protected from all lateral strains and caused to function in its normal balanced posture without artificial support of the arch. None of the joint movements of the foot are in any way interfered with, and no compression is produced upon any of the important plantar structures, since there is no contact with the under surface of the foot except at the heel (Fig. 16).

There exist wide differences of opinion in regard to the use of arch supports. They should be regarded as "splints" and as such are of great value in the treatment of foot disorder. Their action is very positive when they are properly fitted, but prolonged use of the arch supports, like the use of a splint in any other part of the body, leads to a weakening of the structures by supplanting normal function, restricting joint movement, and obstruction of circulation by compression of the important plantar vessels and soft structures. Consequently, their use must be properly supervised and always in conjunction with special exercises to improve and maintain the general condition of the tissues.

Other useful measures to be employed to advantage during the periods of acute or sub-acute subjective symptoms are intervals of rest, preferably with the feet elevated in order to overcome any sluggishness of circulation, and alternate hot and cold foot baths. Massage is always beneficial, but not as important as rest and relaxation during the more acute stages.

When appreciable changes have become established in the bones, although a corrective effort is indicated in order to restore, if possible, a structural balance of the foot, permanence of improvement is largely a matter of careful and prolonged treatment and of the degree to which those changes have taken place. In these cases treatment is essentially palliative, but naturally with the hope that more may be accomplished.

In advanced cases of deformity operative procedure may be indicated but should be regarded as an extreme measure and approached with careful deliberation. The wide dissemination of the deformity throughout the many bones in the foot makes such operations very difficult and of uncertain ultimate results. Many have been devised, tried out, and discarded as unsatisfactory. Lengthening of the heel cord is almost invariably indicated during the course of operation on such cases and is often clearly indicated in milder types of the disorder which present an acute shortening of the calf muscles.

LOOSE BODY FORMATION IN SYNOVIAL OSTEOCHONDROMATOSIS WITH SPECIAL REFERENCE TO THE ETIOLOGY AND PATHOLOGY.*

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THE formation of synovial osteochondromas is a rare pathologic process which has been found in various joints, bursæ, and tendon sheaths. The term "osteochondromatosis" is limited to those synovial osteochondromas unassociated with frank osteoarthritis or synovitis. For some undiscovered reason, the synovial membrane in cases of osteochondromatosis forms bodies which contain either cartilage or bone, or both cartilage and bone. At first, the bodies are attached to the synovial membrane by pedicles, but these pedicles may easily be broken and thus allow the bodies to wander about in the joints, bursæ, or tendon-sheaths. Whether bodies derive nourishment from the synovial fluid and increase in size after detachment has not been conclusively established. The formation is best considered a benign neoplasm, for the absence of metastases is an outstanding feature. The process is peculiar in that the bodies are formed from the synovial membrane instead of from the articular surface, as in the case of osteochondritis dissecans and at times in osteoarthritis. The bodies formed in synovial osteochondromatosis are composed of organized tissues, and are distinct from those bodies that are composed of unorganized tissues, and which are termed "corpora oryzoidea", or "rice-bodies".

REVIEW OF THE LITERATURE.

In reviewing the literature, it has been found that synovial osteochondromatosis has not been well catalogued, and it has been necessary to cull many cases from those indexed merely as loose bodies. However, certain authors have attempted to give descriptive names to the process: such as, synovial chondromata, osteochondromatosis, joint chondroma, chondromatosis of the joint-capsule, and diffuse enchondroma of the joint-

* Thesis submitted to the faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Orthopedic Surgery, November, 1923.

capsule. Besides the twenty-seven case reports, there have been numerous references by various authors to cases that they have observed, but of which complete reports are lacking.

Since 1558, when Ambrose Paré incised a knee-joint and released a loose cartilage, various observers have manifested an exceptional interest in these joint-bodies. Credit for the first description of the formation of joint-bodies by the process under discussion has been given to Laennec, who wrote in 1813. Laennec believed that the bodies originated in the subsynovial tissues. Brodie, in 1836, believed that the bodies arose on the external surface of, or within, the substance of the synovial membrane, because the synovial membrane could be seen to be reflected over the bodies before they had become detached. It is interesting to note that Brodie's conception of the etiology agrees closely with our ideas at the present time. It had been generally believed that loose bodies originated from coagulated lymph, effused from the synovial membrane, by a process of vascularization of the lymph coagulum. However, in a majority of his cases, no symptoms of inflammation had preceded their formation; and thus, at least in some instances, he believed it probable that they arose "like other tumors, in consequence of some morbid action of a different nature."

Synovial osteochondromatosis was known to Macartney, who wrote in 1841. In an article, "On the Movable Cartilaginous Bodies Found in the Synovial and Serous Cavities," he said: "It is a very generally received opinion amongst surgeons that these substances are found in the first instance on a peduncle or footstalk, by means of which a vascular union is maintained between them and the surface of the cavities in which they are enclosed, and that it is only by the accidental rupture of this footstalk that they are set free." He thought it undoubtedly true that such was the case at times, for bodies had been found attached by peduncles to joint-surfaces, to the tunica vaginalis, to the pleura, and to the peritoneum. Evidently he did not have a clear conception of the process, for he thought that the pedicles were composed of "coagulable lymph." However, he knew that the pedicles contained blood-vessels. He mentioned instances of isolated bodies which seemed to have developed without vascular connection.

In 1848, Rainey described the formation of osteocartilaginous bodies by the synovial membrane. He was of the opinion that the origin of the joint-bodies, as well as the fact that they were found loose within the joints, could be explained by studying the synovial membrane. He wrote: "In those parts of a joint, theca, or bursa, where it is least exposed,

it (the synovial membrane) consists of loops and convolutions of capillaries of various degrees of complexity, proceeding from the surrounding vessels and projecting into the synovial cavity; these capillaries are enclosed in sacculi of basement-membrane, studded with minute oval cellules. From the sacculi endowing the capillaries, numerous other sacculi into which no capillaries enter, proceed. These are of various forms and sizes, but generally are attached to the primitive sacculus by an extremely long and slender filament of fibrous tissue, resembling the petiole of a leaf, the secondary sacculus being its expansion. . . . In every instance the secondary sacculi are extravascular. Now this being the apparatus by which synovia is elaborated in all parts in which this fluid is found, and the bodies thus described being found in these situations, they may be inferred to be the product of disease in these structures; the cellules of those fringes in the place of elaborating synovia from the blood, producing, under the influence of morbid action, other products—such as cartilage, which becomes converted into imperfectly formed bone. The fact of the secondary sacculi being connected to the primary by extremely slender pedunculi, will suffice to explain the reason why those bodies may become formed in the first instances, the pedicle serving both to keep them attached and to convey the material from the blood necessary for their development until they acquire a certain size; but afterwards from its tenuity becoming no longer capable of holding them, it breaks and the bodies become loose and most likely cease to enlarge."

Rokitansky* believed that the bodies originate in the subserous cellular tissue, or in the serous tissue itself, and reach the joint-cavity by forcing inward the synovial membrane, remaining attached by pedicles for a time, and then becoming loose by rupture of the pedicles. In 1851, he held the theory that this was the only method by which loose bodies were formed. Virchow, in 1863, had adopted Rokitansky's view to a certain extent.

Kölliker, in 1853, shed more light on the subject in his description of the appearance and structure of the synovial membrane. He described the vascular processes of the synovial membrane that "are met with in almost every joint, constituting, provided that the blood-vessels are filled, red, flattened projections of the synovial membrane, with an indented and plicated margin, and furnished with minute processes. These folds are usually placed close to the junction of the synovial membrane with the

* Rokitansky's Pathological Anatomy was published in 1842. Additional volumes appeared between 1842 and 1846. The English translation of the third volume was published in 1850.

cartilage, upon which they lie flat, thus forming in many cases a sort of coronal around it; in others they are more isolated and placed in other parts of the articulation. In their structure they differ from the rest of the synovial membrane, principally in their great vascularity, consisting as they do of little else than minute arteries and veins, and delicate capillaries forming many loops at the edge of the processes, and consequently they are very similar to the choriod plexuses in the ventricles of the brain. Besides the vessels, they present a matrix of frequently distinctly fibrous connective tissue, the usual epithelium of the synovial membrane, occasionally solitary or numerous fat-cells, and, more rarely, isolated cartilage-cells. At the edge, they are almost invariably furnished with minute foliated conical membranous processes of the most extraordinary forms (often resembling the stems of a cactus), which also frequently contain vessels, but are for the most part constituted merely of an axis of indistinctly fibrous connective tissue, with occasional cartilage-cells, and an epithelium, very thick in places." He stated that it is the avascular appendages of the vascular processes that give rise to the loose bodies, first by gradual enlargement and then by detachment. "These bodies," he said, "are also met with in mucous bursæ and the sheaths of tendons, . . . and they are not developed externally to the synovial membrane, but from any outgrowth of that membrane itself."

Marsh was of the opinion that some, if not most of the loose cartilages, arise "by abnormal growth of fine processes of the synovial membrane." He described two museum specimens that illustrated the process. The first was a shoulder-joint, the inner surface of whose synovial membrane was studded with many club-shaped processes. Some of them consisted of fat and blood-vessels enclosed in a basement-membrane, while the largest ones were found to be fibrocartilaginous in structure. The second specimen was that of an elbow-joint. A pear-shaped nodule of fibrocartilage, the size of a small bean, was found attached to the margin of the greater sigmoid cavity of the ulna by a pedicle, one-third of an inch long and the diameter of a fine string.

Various other observers speak of this process of loose cartilage formation: Dick, in 1866; Barwell, in 1876; and Kirmisson, in 1886. Humphry, Professor of Surgery at Cambridge, in 1888 seemed impressed by the relation of trauma to the phenomenon. He thought that a local irritation, as a sprain, wrench, or blow, might produce villous outgrowths. He believed that these, in turn, came to contain cartilage and perhaps bone, became pedunculated, and then detached to form loose bodies.

In 1888, Kirmisson reported a case, the operation having been per-

formed by Krug-Basse. Ten bodies had been removed from the knee-joint, and all except one were firmly adherent to the serous membrane by filaments.

Because the majority of observers, following Rokitansky, had thought that the finding of occasional cartilage-cells in the synovial villi was a basis for the explanation of all joint-bodies, Patteson, in 1890, stressed the point that an exaggerated rôle should not be assigned to the synovial membrane.

Halstead, writing in 1895, attributed some importance to the relation of inflammatory changes in the joint to the origin of synovial chondromas, for he said that Volkmann had shown that a proliferation of cartilage-cells takes place in the ends of the swollen villi in most chronic inflammations of the joint.

Bolton, in 1896, also spoke of bodies originating in the inflammatory changes in the synovial membrane. His summary of seventy-two collected cases of joint-bodies of various types must have included a number belonging to the group of the synovial osteochondromas, for he mentioned one instance in which synovial membrane was covered by hundreds of cartilaginous bodies.

In the literature of the twentieth century on synovial osteochondromatosis, the report of Reichel comes first. He reported very completely a case in which the knee-joint was involved (Table 1, Case 1). He mentioned the fact that the occurrence of cartilage-cells in synovial villi was known and also that they grew to a rather large size in chronic inflammatory processes of the joint. He considered that his case was exceptional because the inflammatory process was entirely lacking and because the growths were confined to the synovial membrane while the joint surfaces were free. He believed that his case was the first on record, although he credited Müller with a report of a case at a surgical meeting in 1898. In considering the etiology, Reichel stated that he could hardly free himself from the impression that some contagious agent had gotten into the joint. He considered the condition non-malignant. Müller's case appeared in the literature in 1902. A metacarpophalangeal joint was involved, and the origin of the tumor seemed to be at the juncture of the capsule and the periosteum.

The next article was by Riedel, in 1903. He reported the occurrence of the process in the ulnar side of the wrist.

Langemak, in 1904, described the involvement of the talonavicular joint in the tarsus. The head and neck of the talus were involved in the cartilaginous growth, and there were multiple tumors over the inner surface of the joint-capsule. He believed that the epiphysis in the anterior part

Table 1

Cases reported from the literature (1900 to 1923)

Case	Author	Nationality	Year reported	Age	Sex	Joint affected	History of trauma	Duration of symptoms years	Remarks
1	Reichel	German	1900	36	M	Knee	No	5.0	
2	Müller	German	1902	25	M	Metacarpo-phalangeal	No	0.75	
3	Langemak	German	1904	17	M	Talo-navicular	Not mentioned	0.50	
4	Lexer	German	1907	27	M	Knee	No	5	
5	Riedel	German	1907	22	F	Wrist	No	5	
6	Lichtenberg	German	1907	?	?	Knee	?		
7	Marsh	English	1910	19	M	Knee	Yes	?	Severe trauma six years before
8	Kobylinski	Russian	1910	56	M	Knee	Not mentioned	0.66	
9	Rehn	German	1911	28	M	Elbow	No	9.5	Trauma was probably not a factor
10	Bibergeil	German	1913	18	M	Carpus	No	1.5	
11	Hagemann	German	1914	25	F	Shoulder	No	3.0	
12	Eden	German	1914	22	M	Hip	No	0.25	
13	Kobylinski	Russian	1914	?	?	Elbow	?	?	
14	Troell	Swedish	1914	26	F	Knee	Not mentioned	1	
15	Carothers	American	1914	56	M	Both knees	Yes	7	Bilateral involvement
16	Henderson	American	1916	40	M	Knee	Yes	5	Severe trauma twenty-two years before
17	Kopp	Dutch	1916	25	M	Knee	Questionable trauma	4	
18	Kopp	Dutch	1916	39	M	Both knees	Yes	16	Trauma nineteen years before
19	Henderson	American	1917	26	M	Knee	Yes	10	Severe trauma ten years before
20	Kienböck	German	1917	48	M	Ankle	No	7	Similar to Case 18, Table 2.
21	Henderson	American	1918	26	M	Elbow	Yes	5 to 6	Trauma thirteen and six years before
22	Hahn	German	1919	45	M	Knee	Not mentioned	7	Necropsy specimen
23	Lotsch	German	1920	35	F	Knee	Not mentioned	4	
24	Lotsch	German	1920	47	F	Knee	Not mentioned	2.5	
25	Fisher	English	1921	31	M	Knee	Not mentioned	6	Case of Sir Thomas Smith
26	Fisher	English	1921	14	F	Knee	Not mentioned	2	Case of Mr. Harrison Cripps.
27	Ziegner	German	1921	*	M	Elbow	?	?	

*Young soldier.

of the talus was the point of origin. Hagemann later took exception to this, pointing out that the tarsal bones have no epiphyses. In the light of subsequent observations it is reasonable to group this case with those of synovial osteochondromatosis.

Livingston, in an article published in 1906, quotes the theory of Gwilym Davis to the effect that the synovial fringes become inflamed as a result of injury and thus develop the bodies.

Lexer, in 1907, presented a case in which the knee-joint was involved. The case was exceptional in that the bodies had ruptured the capsule and were found in the nearby muscles. The tibia had also been invaded by this growth. However, the growth had spread by pressure rather than by infiltration. Building his theory on the teachings of Virchow and Ribbert, Lexer held that this chondroma, like all other chondromas, originates from scattered embryonic rests, which have arisen by a fault in the mesenchymal differentiation in the formation of the joint. He pointed out the intimate developmental relationship of synovial membrane to cartilage.

Another case of synovial osteochondromatosis of the knee-joint was reported by Lichtenberg in the same year, and still another by Kobylinski in 1910.

In the third edition of Marsh and Watson's book on "Diseases of the Joints and Spine" (1910), there is found the report of a case by Marsh, who, twenty years before, had removed thirty bodies from the knee-joint and five from the semi-membranosus bursa. Some of the bodies were still attached to the synovial membrane by slender pedicles. In the same chapter there is a report of a case in which 415 bodies were removed from the knee-joint by Sir Thomas Smith.* Some of these bodies were still attached.

Rehn, in 1911, reported the involvement of an elbow-joint. The trauma that was mentioned could not have been an etiologic factor, for three days after the injury there appeared a tumor the size of a cherry.

Bibergeil reported the next case in 1913. The ulnar side of the wrist-joint was involved.

A very interesting and instructive case was reported by Hagemann in 1913. Besides diffuse involvement of the synovial lining of a shoulder-joint, there were osteocartilaginous masses in the bursa beneath the coracoid process and in the synovial sheath of the tendon of the long head of the biceps muscle. Each of these pockets was connected with the cavity of the shoulder-joint by a small opening. Hagemann believed that the masses had developed within these sacs, rather than that they had wandered in from the joint-cavity, because of the very small connections

* Second case reported by Fisher (Table 1, Case 26).

with the joint-cavity and because these masses were the oldest and most advanced in calcification. He thought it unlikely that these older tumors could be secondary growths. He was anxious to establish the fact that these masses had developed *in situ*, for this would indicate that the cartilage or bone of the articular structures had nothing directly to do with the origin of the osteochondromas. Since the buræs have neither cartilage nor bone in their structure, it would be reasonable to believe that the masses of cartilage originated in the synovial membrane. Hagemann also observed that the chondromatous growths within the joint-cavity encroached upon the margin of the articular cartilage of the head of the humerus. He used this fact to support, rather than to weaken, the theory that the synovial membrane was the origin of the tumor. He pointed out that at the fourth month of fetal life the margin of the joint surface is covered by vascular and cellular tissue, which is considered to be a prolongation of the synovial membrane. Normally, this tissue disappears, but Hagemann believed that some of it had remained in his case, and had shown the same changes as the lining of the capsule.

Whitelocke sought to explain the development of synovial osteochondromas by a theory based on the changes in the synovial membrane brought on by chronic synovitis occurring in osteoarthritis, or resulting from repeated trauma. He believed that a vicious circle developed when the membrane became thick, edematous, and papillated, while the joint surfaces became rough; for then movement produced more irritation, and irritation produced in turn more pronounced changes in the synovial membrane. Thus chronic irritation of the fibrous elements would produce changes to cartilage and even to bone, which he did not consider strange since all the tissues involved came embryologically from the same group of mesenchymal cells.

Eden, in 1914, reviewed the German literature beginning with Reichel's report in 1900. He reported the involvement of a hip-joint. Agreeing with other German observers, he believed that trauma and infection could be excluded when considering the etiology. He claimed that by microscopic examination he could see the stages of transformation from synovial cells to cartilage-cells. In his article Eden mentioned the fact that a second case had been reported by Kobylinski. The process occurred in the elbow-joint in this instance.

Troell, of Stockholm, reported in the same year a case of synovial osteochondromatosis of the knee-joint. He also reviewed the German literature dating back to 1900. He believed that he was dealing with a new growth, a tumor of the joint-capsule. Another report, in 1914, was

that by Carothers of Cincinnati. The case that he reported was noteworthy in that both knee-joints were involved. Previous to this report, mono-articular involvement had been stressed as a constant feature. In this case, however, many bodies were removed from each knee-joint. The three etiologic factors of exposure, trauma, and infection were considered. The patient, a negro, had been a longshoreman for many years and had been accustomed to lifting heavy objects on his thighs while in a stooped position. Infection was considered the most obvious factor, for the man had had both gonorrhea and syphilis. In considering the infectious factor, the theory was advanced that the bacteria had localized near the joints, producing mild chronic irritation; this in turn caused chronic perivascular changes which brought about pressure on the blood-vessels, with consequent chronic congestion, hyperplasia, and edema of the fibrous tissue. In this manner the villous arthritis was accounted for, and traumatic hemorrhage into the villi was believed to be the basis for the calcification.

In an article published in 1915, Heineck reviewed the cases of joint-bodies of all types reported in the English, French, and German literature between 1890 and 1913. In this article he mentioned the process of joint-body formation, which we consider synovial osteochondromatosis. Following Kölliker, he spoke of the occasional occurrence of cartilage-cells in the fringes of the normal synovial membrane. He thought that morbid conditions, sprains, irritations, and so forth could act as stimuli to cause the cartilage-cells to divide and to multiply with the ultimate production of loose joint-bodies.

In 1916, Henderson reported his first case. This is reported in detail below as Case 1, in the series reported from the Mayo Clinic. In the same year Kopp, of Holland, reported three cases of loose body formation in knee-joints. Two of these can undoubtedly be classified as synovial osteochondromatosis, but there is some question about the third case. Apparently there was no operation performed in the first case, but the roentgenographic appearance is so typical that the diagnosis of osteochondromatosis must stand. The diagnosis in the second case was substantiated by operation. Bodies were found in the supra-patellar pouch as well as in a bursa posterior to the knee-joint. This second case is of extraordinary interest because the opposite knee was also involved. However, the second knee was not explored. There is a chance to doubt the diagnosis of bilateral osteochondromatosis in the third case. The roentgenograms were not so typical and the diagnosis was not corroborated by an operation. Kopp believed that the small bodies started as osteomas, because he found that the smallest ones were composed of bone.

Table 2.

Cases from the Mayo Clinic (1910 to 1923)

Case	Joint affected	Age	Sex	History of Trauma	Number of bodies	Duration of symptoms before examination, years	Remarks
1	Knee	40	M	Yes	Multiple	17	Associated chondrosarcoma lower end of femur.
2	Knee	26	M	Yes	25	10	
3	Knee and bursae	50	M	No	104	3	
4	Bursa beneath gastrocnemius	62	M	No	4	25	
5	Bursa	29	M	?	1	4	
6	Both knees	60	F	No	8	12	
7	Knees and bursae	50	M	No	14	5	
8	Knee	49	M	Yes	Very many	4	Trauma twenty-nine years; "laid up three weeks"
9	Shoulder	20	F	No	10	12	
10	Elbow	37	M	No	31	4 to 5	
11	Elbow	26	M	Yes	65	13	
12	Elbow	24	M	No	107	9 to 10	
13	Elbow	23	M	No	21	0.83	
14	Elbow	66	F	Yes	3	12	Fall twelve years before.
15	Flexor tendon-sheath of finger	33	M	Yes	5	3.5	Severe trauma four years before
16	Knee	28	M	No	200	18	Thirteen years before, threw elbow out of joint.
17	Ankle	28	M	?	14	4	Bumped ankle four years before.
18	Elbow	30	M	Yes	125	3.5	
19	Elbow	26	M	Yes	12	7	Fall seven yearsbefore

Henderson's second case of osteochondromatosis of the knee-joint was reported in 1917. At that time he spoke of trauma as a possible factor and thought it reasonable to consider the formation a non-malignant new growth. The case is reported in detail as Case 2 of this series. In the same article Henderson mentioned that he had twice observed the condition in the elbow-joint. He reported the involvement of an elbow-joint in 1918, and the detailed case history may be found in Case 11 of the series reported below.

Kienböck, in 1917, reported four cases which he diagnosed chondromas of the joint-capsule. However, only one of these can safely be classified as a case of synovial osteochondromatosis. No operation was performed in the first case and no microscopic examination was made in the other two cases. In the fourth case the ankle-joint was as large as two fists. At operation the joint-cavity was found to be distended by chondromatous masses which were separated by fibrous septa, and showed the changes of calcification and ossification. He said that the origin was manifestly the capsule, most of which had been destroyed by the tumor; and he thought that the point of origin was probably at the zone where the capsule was attached to the bone. This case closely resembles Case 18 of the reported series.

Hahn, in 1919, in an article dealing with the origin of joint-bodies in arthritis deformans, published a group of eight cases in which outspoken arthritis deformans was associated with loose body formation. Among these cases was one in which the bodies evidently arose as synovial osteochondromas in the knee-joint. A very detailed description was given of the gross and microscopic appearance of the loose bodies as well as of a series of vascular villi, each of which contained a small spherical shell of bone filled with fat. The small bodies in the villi resembled the large detached bodies. This case would not be classed as true osteochondromatosis because of the frank arthritis.

Contributions to the literature of 1920 were made by Lotsch and Kappis. Lotsch reviewed the cases reported in German since 1900 and added two cases, in each of which the knee-joint presented the typical picture of synovial osteochondromatosis. Kappis discussed the condition, but did not report a case.

Fisher, writing in 1921, divided his consideration of synovial chondromas into those cases associated with osteoarthritis and those cases not associated with osteoarthritis. He said that the two types presented the same gross and microscopic appearances. He reported three cases in which the knee-joint was involved. However, the first two of these had

already been reported by Marsh. Marsh, Sir Thomas Smith, and Harrison Cripps were the surgeons in the three instances. Fisher believed that the etiology was bound up with the etiology of tumors. He spoke of the relationship that the various tissues bear to each other embryologically, and considered that trauma was undoubtedly a contributing factor.

The most recent case found in the literature was that reported by Ziegner in 1921. The patient was a young soldier whose elbow-joint was involved. In the same year the condition was mentioned by Lamson, but a case of osteochondromatosis was not reported in the article.

REPORT OF CASES OBSERVED AT THE MAYO CLINIC.

Case 1 (A 131188). Osteochondromatosis of the right knee-joint associated with chondrosarcoma of the distal end of the right femur. May 19, 1915, the patient, a farmer, forty years of age, came to the Clinic complaining of stiffness and pain in the right knee, with great increase in size. Twenty-two years before, he had fallen and had broken a bone in the right leg near the knee. He had recovered from this in about a month. About five years later the right knee began to swell just above the patella and it continued to enlarge. Enlargement had been especially rapid during the three to six months preceding examination. Pain had been worse for three months.

The patient, a well-developed man, walked with a limp. There was an enlargement of the right knee and the region of the thigh just above the knee. The right knee-joint moved from about 170 to 90 degrees. The roentgenogram (Fig. 1) revealed multiple calcified bodies in the knee-joint, a large area of destruction in the lower end of the femur, and a small exostosis on the medial aspect of the lower end of the femur.

May 20, 1915, a small tumor was removed from the left tonsil and found to be a fibroma. May 27th, the right knee was explored by a median incision through the patella and multiple loose bodies were removed. Besides many small bodies, there was one which measured 2.5 by 0.6 cm. The bodies clearly had their origin in the synovial membrane. The exostosis within the joint over the lateral condyle was removed. The lower end of the femur was explored through a medial incision and a tumor found which appeared benign clinically. The pathologist's report was chondroma.

The growth in the femur recurred, and on September 23, 1915, amputation was performed in the middle third of the right thigh. The

pathologist's report at this time was chondrosarcoma of the lower end of the femur. Microscopic sections showed in certain regions a cellular tissue in which mitotic figures were numerous. In other areas the sections revealed actively growing bone and cartilage, as well as a great deal of fibrous connective tissue. The osteochondromatosis did not recur in the joint.

FIG. 1 (Case 1).

Osteochondromatosis of knee-joint associated with chondro-sarcoma of the femur; (a) multiple loose bodies; (b) exostosis; (c) sarcomatous degeneration.

Roentgenograms of the chest on December 15th revealed the presence of multiple metastatic growths in the lungs. A letter from the attending physician stated that the lung condition progressed. The patient died March 5, 1916.

Case 2 (A 157963). Osteochondromatosis of the left knee-joint. April 21, 1916, the patient, an oil driller, twenty-six years of age, came to the

Clinic complaining of locking in the left knee. Ten years before, the knee had been injured so severely that swelling had persisted for six months. Since that time the patient had been bothered by a locking in the joint every month or so. The locking had become more frequent and more painful. He had had over 100 attacks and recently had been disabled for a week at each time. No other joints were involved and his general health had always been good. Aside from slight limitation in flexion of

FIG. 2 (Case 2).
Osteochondromatosis of the knee-joint.

the left knee, the examination was essentially negative. The roentgenogram (Fig. 2) revealed shadows of multiple calcified bodies in the joint and also extending up the anteromedial aspect of the distal part of the femur.

April 27, 1916, nineteen loose osteocartilaginous bodies were removed from the anterior compartment of the knee-joint (Fig. 3). The bodies clearly arose from the synovial membrane, which was pouched and marked by fibrous tags, some of whose tips were cartilaginous. June 23, 1916, six loose bodies were removed from the posterior compartment of the joint.

Microscopic sections of one of the small attached bodies showed that it was composed of vascular connective tissue surrounding a spherical shell of bone which was filled with fat. The bone did not appear to have been proliferating rapidly, although there was a capillary here and there. Most of the blood supply had probably been shut off from the bone, and that which remained had not given sufficient nutrition to cause the bone to grow. Also it is possible that the supply had been entirely cut off, and then partially re-established. A section of one of the larger loose bodies

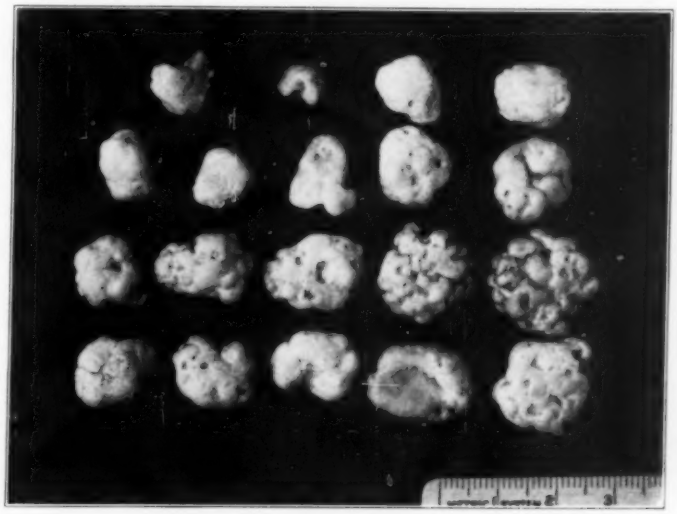

FIG. 3 (Case 2).
Nineteen loose osteocartilaginous bodies removed from the knee-joint.

showed that it was composed almost entirely of hyaline cartilage. Around the periphery there was a fibrous tissue whose cells were arranged parallel to the surface. Beneath this came cartilage with rows of cells parallel to the surface. Deeper within the body, the cells tended to be arranged in rows perpendicular to the surface. Some of the more superficial cells showed that they had been well preserved, for they filled the cell spaces in the matrix and the nuclei stained well. The cartilage deeper within the body was necrotic, for there were wide spaces in the matrix around each cell and the nuclei were pyknotic or karyolytic. At the center of the body there was a quantity of formless, eosinophilic, necrotic material as well as a small cavity.

Case 3 (A 201151). Osteochondromatosis of the left knee and bursæ about the knee, with similar involvement of the right knee. The patient, a farmer, fifty years of age, was first examined at the Clinic, April 21, 1916. For three years he had noticed that flexion caused pain in the knee. Two years before, he had first noticed a "bunch" posterior to the knee; this had grown gradually.

On examination a prominence was found on the anteromedial aspect of the left knee and a hard mass in the left popliteal space. The roentgenograms revealed multiple mulberry-like shadows in the vicinity of the left and also the right knee.

September 15, 1917, and again March 11, 1920, a great number of loose bodies were removed from the bursæ and joint-capsule of the left knee. A chondroma was removed from beneath the popliteus muscle on the posterior surface of the proximal part of the left tibia. The extra-articular bodies were found within definite sacs lined with synovial membrane, and it was considered that they were probably abnormally placed bursæ. The synovial membrane of the joint showed many very small, pedunculated cartilaginous bodies.

Case 4 (A 239850). Osteochondromatosis of bursa beneath right gastrocnemius muscle. July 26, 1918, the patient, a farmer, sixty-two years of age, came to the Clinic because of a "growth" back of the right knee. He had noticed this for ten years. For twenty-five years he had had slight attacks of trouble in the knees which he called "rheumatism." Palpation disclosed a tumor about the size of a hazel nut in the right popliteal space. It was slightly nodular, suggesting the presence of loose bodies outside the joint proper. Movement of the knee-joints caused some discomfort. A roentgenogram of the right knee revealed multiple calcified bodies posterior to the condyles.

August 1, 1918, seven bodies were removed from the bursa under the right gastrocnemius muscle. The four larger bodies measured, respectively, 2.2 cm., 1.9 cm., 0.9 cm., 0.6 cm. in diameter. These bodies were believed to have originated from the lining of the bursa, for there were a few small bodies not more than 1 mm. in diameter which were growing from the synovial membrane.

Case 5 (A 243018). Osteochondromatosis of the right knee. August 21, 1918, the patient, a lumber retailer, came to the Clinic because of soreness in the right knee as well as the sensation of something slipping within the joint. In 1914, he had twisted the knee while scuffling. It had "burned" somewhat at the time, and one month later he noticed, lateral to the patella,

a swelling the size of a hickory nut. The body had been felt but could not be located at the time of examination.

August 29, 1918, the right knee-joint was opened and a piece of cartilage 2 by 1.5 by .5 cm. was found lying loose in the intercondylar notch. Lying about on the synovial membrane, and attached to it in most instances, were small flakes of cartilage about 2 mm. in diameter. Several of the large flakes and a few of the small ones were removed in addition to

FIG. 4 (Case 7).
Roentgenogram of the right knee. Note cauliflower-like appearance of bodies.

an area of synovial membrane which contained six or eight of the small bodies. The pathologist reported that the large body was composed of cartilage.

Case 6 (A 41317). Osteochondromatosis of the right knee-joint. June 26, 1920, the patient, a woman, sixty years of age, came to the Clinic. Since 1908 she had been troubled by aching pains in the knees, worse in the right knee. At times the pain was accompanied by a feeling that something had slipped within the joint. In 1910, at operation at the

Clinic, a large number of gallstones were found and cholecystectomy was therefore performed.

Roentgenograms of the right knee revealed five definite bodies in the suprapatellar region, and roentgenograms of the left knee, two bodies.

July 10, 1920, four large and four small bodies were removed from the right suprapatellar pouch. Some of them were still attached to the syno-

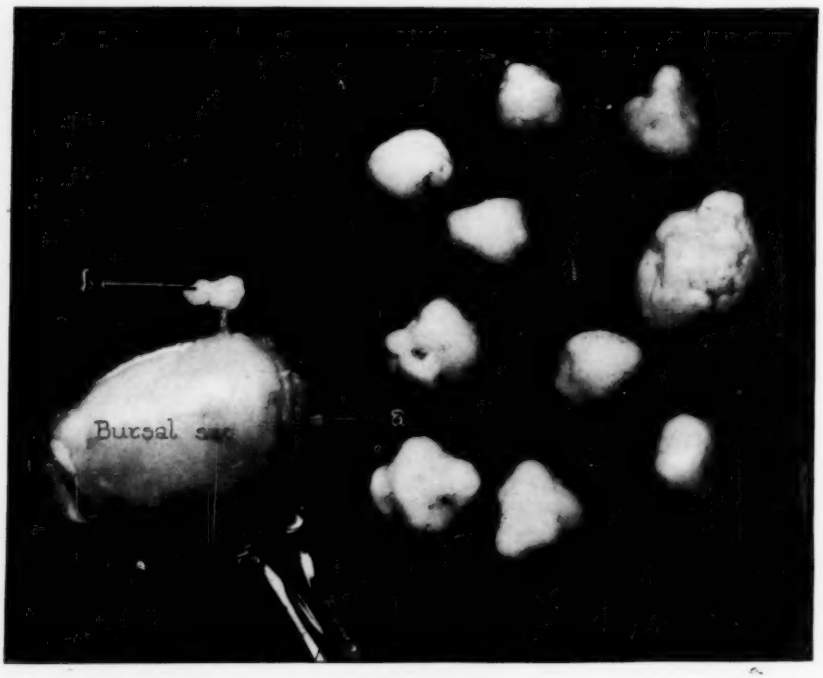

FIG. 5 (Case 7).

Bursa from beneath medial head of right gastrocnemius with ten loose bodies which it contained; (a) vascular hypertrophic villus; (b) pedunculated osteocartilaginous body. The bursa was inverted and stuffed with cotton.

vial membrane by fibrous pedicles. The bodies were not removed from the left knee because they were not causing symptoms.

Case 7 (A 346313). Osteochondromatosis of both knees. The patient, a cabinet maker, fifty years of age, registered January 14, 1921. Since 1916 he had been bothered by a "smarting pain" in each knee, when he stood or walked. In 1918, loose bodies had been removed elsewhere

from both knee-joints. After this he was better for a time, but not completely free from the pain which gradually increased. There was no history of locking.

Examination revealed swelling in each popliteal space. Aside from varicose veins of both legs and dental sepsis, the remainder of the physical examination was essentially negative. The roentgenograms revealed multiple calcified bodies posterior to and within each knee-joint (Fig. 4).

January 20, 1921, a bursa containing ten loose bodies was removed from beneath the medial head of the right gastrocnemius muscle (Fig. 5).

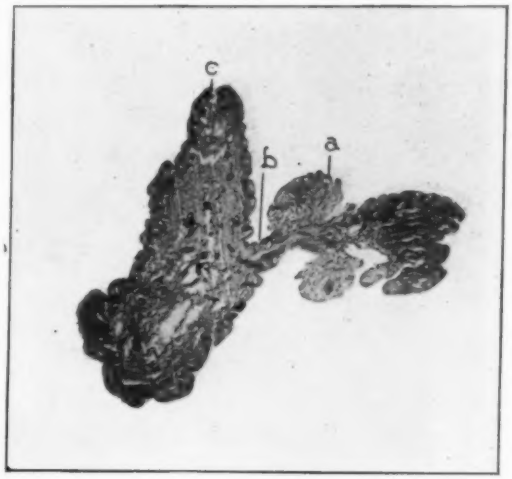

FIG. 6 (Case 7).
Photomicrograph of vascular hypertrophic villus, such as Fig. 5a; (a) vascular villus; (b) vascular pedicle; (c) wall of bursa.

The bodies were pearly white in appearance and possessed the elastic hardness of cartilage. Scattered over the synovial membrane were countless hypertrophic synovial villi which in some areas gave the impression of velvet and in other regions had the appearance of a miniature forest.

From the bursa beneath the medial head of the left gastrocnemius muscle were taken two bodies—one very rough and cauliflower-like, pearly white and hard; the other, darker in color, not so hard, and on section was seen to contain a great deal of fat, giving the appearance of bone marrow. Special attention is called to this body because microscopic section showed that it had a blood supply. It must have been torn from the bursal wall.

The left knee-joint was opened posteriorly and two more loose osteocartilaginous bodies were extracted. They were dirty yellow, showed evidences of wear, and possessed facets that had evidently been caused by pressure.

Microscopic examination of the material obtained in this case revealed a series of interesting processes. A section of such a vascular hypertrophic villus, as (a) in Fig. 5, is presented to illustrate what might have been an early stage in the formation of the bodies (Fig. 6). Microscopically it was found to consist principally of loose connective tissue supporting

FIG. 7 (Case 7).
A succeeding step in the formation of the osteocartilaginous bodies; (a) thrombosed blood-vessels; (b) canalized thrombosis.

a great number of capillaries. These capillaries were especially numerous just beneath the synovial membrane. In one area there was a small number of fat cells, but no cartilage or bone. In a few regions there was a definite lymphocytic infiltration.

Figure 7 shows what may have been a succeeding stage in the formation of a loose body. The tissue contained many vessels, but the vessels contained little blood. The vessel (b) was almost completely occluded by a canalized thrombus, while the vessel (a) had a smaller central thrombus. There was no cartilage, bone, or fat in the section.

A more advanced stage of development is shown by Fig. 8. This section indicated that the structure was roughly a spherical fibro-osteocartilaginous shell filled with fatty tissue and spurs of bone. The pedicle was avascular and hence the body must have been without a blood supply. The vessels in the bursal wall (Fig. 8, d) near the base of the pedicle were engorged with blood. Figure 9 is a photomicrograph of a higher magnifi-

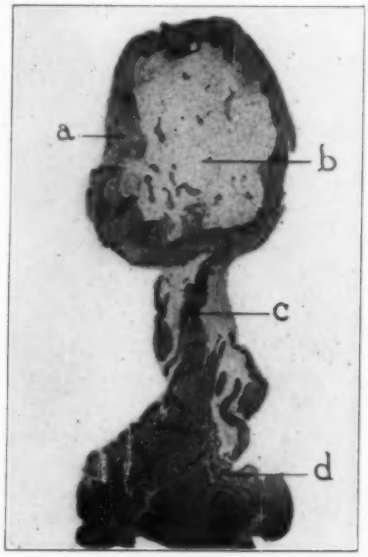

FIG. 8 (Case 7).

Section through bulbous pedunculated osteocartilaginous body (b), in Fig. 5); (a) spherical fibro-osteocartilaginous shell; (b) fatty center with spurs of bone; (c) fibrous avascular pedicle; (d) bursal wall with congested vessels.

cation of a portion of the same section. The fibrous tissue which surrounded the body gradually merged into fibrocartilage. Deeper than this came a zone of calcified cartilage and then a sharp transition to true bone.

It was profitable to study the state of preservation of the various tissues of this body whose blood supply had been shut off. First of all the blood-vessels in the body were found to be thrombosed and necrotic (Fig. 9, f). The cartilage cells appeared to have been well nourished, for

the nuclei were large and the cells filled the spaces in the matrix. The bone-cells had been fairly well preserved; however, the cutting off of the blood supply had produced some changes, for a certain number of the nuclei were pyknotic, and the absence of osteoblasts indicated that active bone formation had ceased. The fatty tissue was fairly well preserved.

FIG. 9 (Case 7).

Enlarged section of Fig. 8; (a) fibrous connective tissue covering body; (b) fibro-cartilage; (c) calcified cartilage; (d) true bone in laminæ, osteoblasts absent; (e) necrotic thrombosed blood-vessels; (f) fatty center.

Figure 10 is a photomicrograph of low magnification of an intra-articular body. Lining the surface were strands of fibrous tissue. Inside of this was found laminated hyalin and calcified cartilage. The central part of the body was composed of necrotic fat and bone. The high-power photomicrograph (Fig. 11) revealed the condition at the center of the same loose body. The necrotic bone-cells showed excellent examples of pyknosis and

karyolysis. Osteoblasts were nowhere to be found. The bone had been laid down in lamellæ (Fig. 11, c). The fatty tissue was almost completely disintegrated. The cartilage at the surface of this body was necrotic as well as that deeper within. The poor condition of the cells near the surface may be explained by the fact that this was one of the intra-articular bodies whose gross appearance showed that it had been subjected to considerable trauma.

In contrast, Fig. 12 shows the hyaline cartilage near the surface of one of the loose bodies found in the bursa. This cartilage was in a better state of preservation than that found near the surface of the intra-articular body. The cartilage deep within this body was very necrotic.

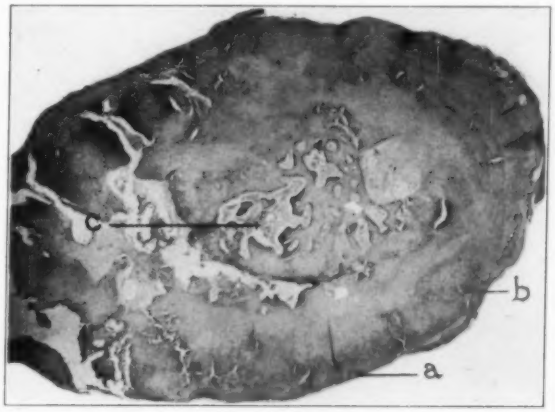

FIG. 10 (Case 7).
Section through an intra-articular body; (a) strands of fibrous tissue on surface; (b) cartilage not well preserved; (c) necrotic bone and fat at center.

In the description of the gross appearance of the bodies special attention was called to a dark body, a section of which gave the appearance of bone marrow. This was the only large body that was found to have a blood supply. The avascular bodies consisted principally of a large proportion of cartilage, a small amount of necrotic bone, and some fat. This dark body which had a blood supply had practically no cartilage in its vascular portion but was composed principally of fat and living bone (Fig. 13). In the vicinity of the blood-vessels the bone was well preserved, as evidenced by the normal appearing nuclei in the bone-cells which filled the lacunæ, and by the presence of osteoblasts.

In looking through a serial section of a region of the synovial membrane which contained many villi, a capillary congestion accompanied by a perivascular lymphocytic infiltration was found. A photomicrograph of an area in one of these sections is shown in Fig. 14.

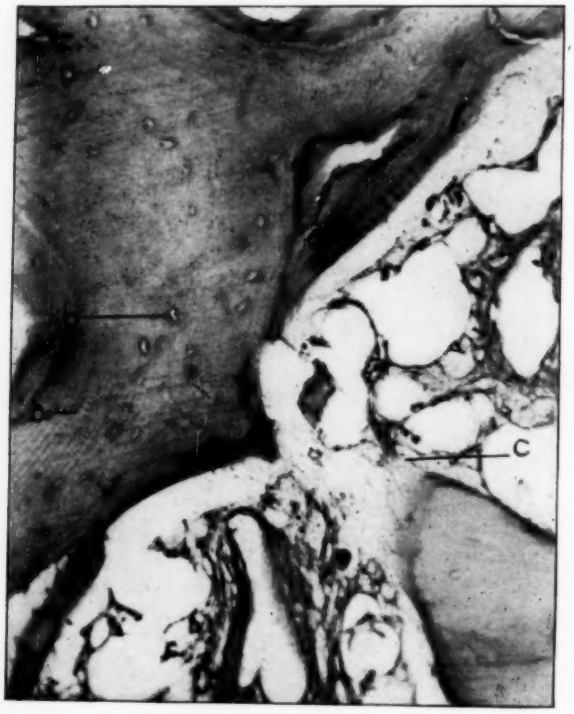

FIG. 11 (Case 7).
Higher magnification of the central portion of Fig. 10; (a) necrotic bone; (b) necrotic fat; (c) lamellæ in bone.

Case 8 (A 114418). Osteochondromatosis of the left knee. October 3, 1914, the patient, a farmer, forty-nine years of age, came to the Clinic because of limitation of motion and swelling of the left knee. Twenty-nine years before, he had been "laid up" for three weeks because of trauma to the left knee. Four years before coming here, swelling began after hard work. Since then there had been a gradual stiffening of the joint, with the

formation of a hard tumor in the popliteal space. There had been no pain and the patient had not been incapacitated.

The roentgenogram (Fig. 15) showed dense deposits of calcium around the left knee-joint with a normal outline of the articular surfaces. The great number of mulberry and shot-like shadows is typical of osteochondromatosis.

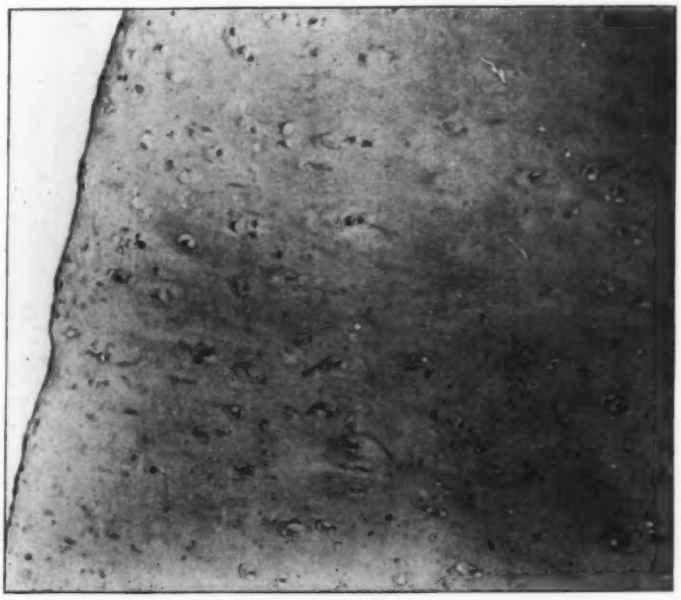

FIG. 12 (Case 7).
Cartilage near surface of bursal body. Note that cartilage is fairly well preserved, also similarity to articular cartilage.

At examination the left knee was found to be about 2 cm. greater in circumference than the right knee. On palpation there could be felt many small hard bodies which gave the sensation of crepitus when squeezed. A hard mass was encountered in the popliteal space. Motion from 170 to 90 degrees was present at the knee-joint. The patient did not submit to operation.

Case 9 (A 238014). Osteochondromatosis of the right shoulder-joint. July 9, 1918, the patient, a woman, twenty years of age, came to the Clinic because of aching of the right shoulder and arm. The trouble dated back

for twelve years. She complained mostly of sudden attacks of quite severe pain lasting for perhaps five minutes and followed by a little residual soreness. This sometimes had come on when reaching. The patient could not lift the arm above the head. The roentgenogram showed shadows of multiple calcified deposits in the right shoulder-joint.

FIG. 13 (Case 7).

High magnification of a section near the surface of a large vascular body; (a) capillaries containing blood; (b) osteoblasts; (c) well-preserved bone; (d) fatty center; (e) membrane covering the body.

July 18, 1918, ten loose bodies were removed from the right shoulder by a posterior incision. The site of origin was not determined at the time of operation because the incision was small. The bodies were manipulated until they could be removed with a gall-bladder scoop. The microscopic sections of one body showed that it was composed principally of fibrocartilage.

December 5, 1919, a roentgenogram indicated that small bodies had reformed in the shoulder-joint. It was thought best to defer operation. The large number of bodies, their size and consistency, the age of the patient and the appearance of the roentgenogram led to the diagnosis of osteochondromatosis.

Case 10 (A 126099). Osteochondromatosis of the left elbow-joint. March 9, 1915, the patient, a farm laborer, thirty-seven years of age, came

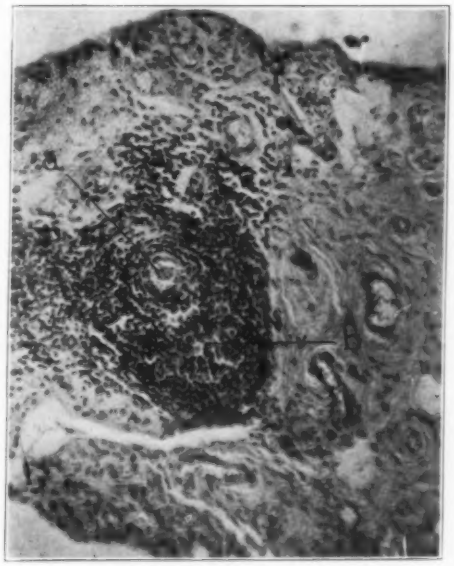

FIG. 14 (Case 7).
Perivascular lymphocytic infiltration found in a hypertrophic villus; (a) vessel; (b) perivascular lymphocytes.

to the Clinic because of limitation of motion in the left elbow-joint which had been gradually becoming worse for four or five years.

Extension of the elbow was limited to about 135 degrees, while flexion was limited to about 45 degrees. The examiner could feel the small bodies. The roentgenogram indicated that there were a number of bodies in the region of the left elbow-joint.

March 18, 1915, thirty-one intracapsular loose bodies were removed from the left elbow by anterior and posterior incisions.

Microscopic examination of a section of one of the bodies showed that it was composed of well-nourished fibrocartilage near the surface, with necrotic and calcified cartilage deeper within.

January 7, 1919, an intra-articular exostosis was removed from the posterior surface of the medial condyle of the right humerus, but no loose bodies were encountered in this joint.

FIG. 15 (Case 8).
Very typical osteochondromatosis of knee-joint. Note regular joint-surfaces.

In spite of the fact that it was not possible to prove the origin of the bodies by inspection of the synovial membrane, the diagnosis of osteochondromatosis was made because of the large number of bodies, their shape and consistency, the appearance of the X-ray plate, and the age of the patient.

Case 11 (A 196125). Osteochondromatosis of the right elbow-joint. The patient, a locomotive engineer, twenty-six years of age, came to the Clinic June 1, 1916, because of swelling and stiffening of the right elbow.

Thirteen years before, he had stuck a pitchfork into the right forearm. Since then he had had difficulty in flexing and extending the elbow. Six years before, he had struck the right elbow on a sharp steel projection, causing intense pain and residual soreness. Later a "lump" developed in the region of the joint, and for five years pain had been a symptom.

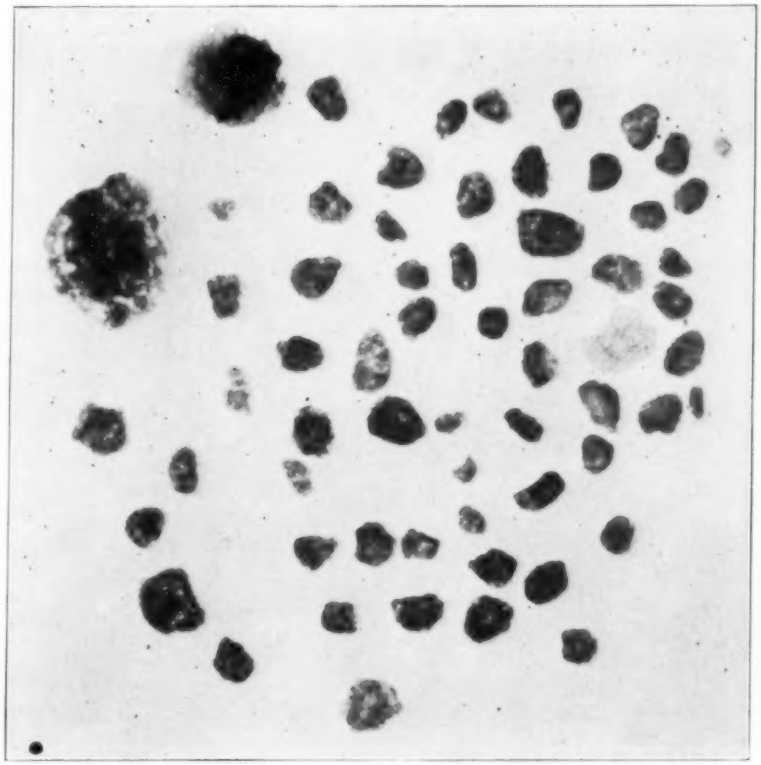

FIG. 16 (Case 11).
Roentgenogram of bodies after removal, showing the fantastic forms in which the calcium was deposited in the cartilage and bone.

Frequent painful locking had taken place for the preceding three years. Forced flexion or extension had especially given pain.

The right elbow was found to be 5 cm. greater in circumference than the left. Extension was limited to about 140 degrees and flexion to about 100 degrees. There was a general enlargment of the lymph nodes, but

otherwise the remainder of the physical examination was essentially negative. The roentgenogram revealed a great number of small calcified bodies in the region of the right elbow-joint, and the roentgenographic appearance was very typical of osteochondromatosis of an elbow-joint.

June 7, 1917, sixty-five rounded loose bodies were removed from the joint. The largest was about 2.5 cm. in diameter, the next largest was

FIG. 17 (Case 12).
Typical lateral view of osteochondromatosis of the elbow.

2 cm. in diameter, and the other bodies were nearly all the size of large peas. There were small cartilaginous tipped structures attached by pedicles to the synovial membrane at several points. The joint showed evidence of chronic irritation.

Microscopic examination of a section of one of the large bodies indicated that it was composed principally of hyaline and fibrocartilage,

which showed various stages of necrosis and calcification in the interior, but signs of progressive changes at the surface. The body was thought to be free, but a few regions of vascular tissue on the surface told that there was probably a connection to the synovial membrane, which had either persisted from the first, or more likely had been re-established after having been broken. There were numerous foreign body giant-cells in and near

FIG. 18 (Case 12).
One hundred seven bodies removed from the elbow-joint.

the connective tissue at the surface. Figure 16 is a roentgenogram taken of the bodies after removal.

Case 12 (A 240416). Osteochondromatosis of the right elbow-joint. July 30, 1918, the patient, a farmer, twenty-four years of age, came to the Clinic because of pain in the right elbow of nine or ten years duration and limited motion of four or five years duration. Two years before, a small

"knot" had appeared posteriorly. All symptoms had been gradually progressive. There had been no redness or other signs of inflammation, and there had been no constitutional symptoms.

Extension of the right elbow was limited to about 100 degrees and flexion to about 60 degrees. The right elbow was enlarged. The roentgenogram showed very typically the multiple calcified bodies of osteochondromatosis (Fig. 17).

On August 1, 1918, 107 bodies, ranging in diameter from seven-eighths to one-sixteenth of an inch were removed from the right elbow (Fig. 18). The attachment of some of the bodies to the synovial membrane indicated the origin from this membrane.

Microscopic section of a body showed that it was laminated. The fibrocartilage of which it was composed was well preserved near the surface, but necrotic and calcified in the deeper layers. No bone was seen.

Case 13 (A 86594). Osteochondromatosis of the right elbow-joint. March 12, 1917, the patient, a farmer, twenty-three years of age, registered at the Clinic. He complained of pain which he had first noticed in the right elbow ten months before, while shoveling dirt. Clinically the joint was negative, except for incomplete extension and moderate tenderness to firm pressure. The roentgenologist reported slight roughening on the anterior aspect of the humerus just above the condyles.

January 3, 1918, the patient returned. For three months he had noticed a swelling about the joint. Pain had continued, especially on use. The roentgenogram at this time revealed multiple joint-bodies.

January 10, 1918, twenty-one loose osteocartilaginous bodies were removed from the right elbow-joint. The site of origin was not determined at this time, but it was thought that they had come from the synovial membrane.

A section of one of the larger loose bodies showed that it was composed principally of necrotic fibrocartilage. The cells near the surface were in a much better state of preservation than those deeper within. At the center there was a cavity surrounded by formless necrotic material.

January 9, 1923, the patient returned. He had gotten along very well until seven or eight months before, when the right elbow had become more painful, especially when used. Active motion was possible from 140 to 45 degrees. Just anterior to the medial condyle of the humerus a movable hard body could be felt.

January 15, 1923, the ulnar side of the elbow-joint was opened anteriorly, and a pedunculated body 7 mm. in diameter was found attached to, and evidently having its origin in, the synovial membrane on the anterior

wall of the capsule. No free bodies could be found. By microscopic examination it was learned that the pedicle was very vascular and that the body had a blood supply. The body consisted of fibrous connective tissue and well-preserved fibrocartilage on the outside, with well-nourished bone at the center.

Case 14 (A 414987). Osteochondromatosis of the left elbow-joint. January 11, 1923, the patient, a woman, sixty-six years of age, came to the Clinic complaining of a tumor of the left elbow. About twelve years before, she had fallen on the left elbow, causing swelling and some pain for a week. Afterwards some swelling remained, but was painless. About November, 1922, it was noticed that the joint was gradually enlarging.

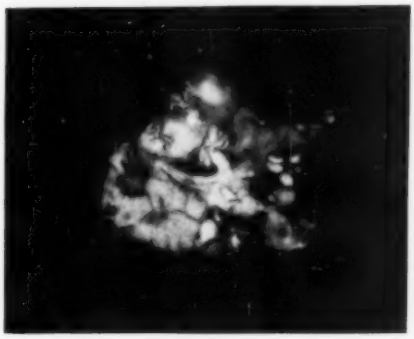

FIG. 19 (Case 14).
Area of synovial membrane, showing pedunculated hypertrophic club-shaped villi.

Enlargement had increased rapidly, with a dull ache but very little pain. Two weeks before, a thin, brown, watery fluid had been aspirated.

In the region of the left elbow was a large fluctuating tumor. There was no tenderness but marked crepitation was felt on motion of the distorted joint. The possibility of osteochondromatosis, or a neuropathic joint, was entertained, but a very careful neurological examination failed to reveal any basis for the latter possibility. The Wassermann reaction was negative. There was a tardy left ulnar nerve palsy from the changes at the left elbow-joint.

The roentgenogram revealed multiple large joint-bodies of the left elbow.

January 18, 1923, the joint was explored. The synovial membrane

showed the typical picture of osteochondromatosis (Fig. 19). Three large loose bodies were removed from the left elbow-joint, the largest being 2.5 cm. in diameter.

Microscopic examination of the sections taken through the small attached bodies shown in Fig. 20 revealed the earliest stage of bone formation that was found in the entire series. The pedunculated hypertrophic villus was found to consist principally of a cellular tissue supporting blood capillaries. Here and there, in the interior of the structure, there were

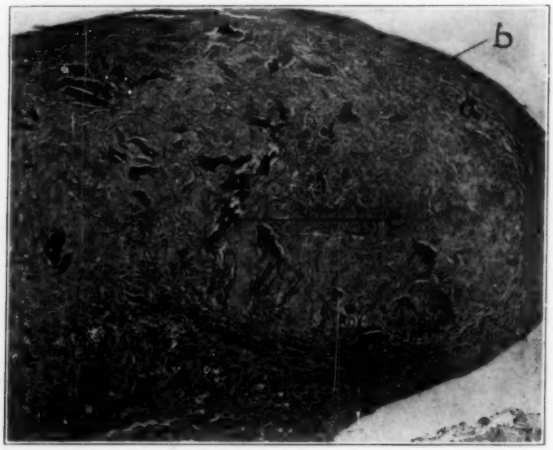

FIG. 20 (Case 14).

Photomicrograph of section of hypertrophic villus shown in Fig. 19; (a) villus; (b) synovial membrane; (c) small particles of bone (stained pink by eosin). Note: another section of a villus in the same case showed a particle of bone such as c, which included a bone-cell in a typical lacuna, with radiating canaliculæ.

found minute spurs of bone with the associated osteoblasts and bone-cells. Certain sections showed numerous foreign-body giant-cells or osteoclasts. Cartilage was nowhere to be found.

Section of one of the large bodies demonstrated one of the typical forms in which these bodies develop. Like the attached body in Fig. 8, it was composed of a spherical shell of fibro-osteocartilaginous tissue filled with fat and spurs of bone. The cartilage was well nourished, but the osseous tissue and fat showed evidences of necrosis. This could be accounted for by the necrotic blood-vessels which contained disintegrating

blood-cells. In many regions there was evidence of hemorrhage. This might have taken place from passive congestion when the blood supply was being cut off.

Case 15 (A 393209). Osteochondromatosis of the flexor tendon-sheath of the middle finger, right hand. The patient, a farm laborer, thirty-three years of age, registered at the Clinic June 1, 1922. About four years before, he had split the middle finger of the right hand and dislocated it backwards by a baseball. It had healed up readily and had seemed all right

FIG. 21 (Case 15).
Five bodies removed from the sheath of the flexor tendon of the middle finger.

for six months. Then the "middle joint" had enlarged and had become stiff and painful at times. About one and one-half years before examination at the Clinic he had noticed, near the base of the middle finger, a hard, movable lump which had been steadily growing. His general health had been good.

Examination showed that the proximal interphalangeal joint of the middle finger of the right hand was markedly enlarged on the radial aspect. Also there was an enlargement between this joint and the second interphalangeal joint. Apparently associated with the flexor-tendon in the

region of the metacarpophalangeal joint of the same finger was a smooth hard lump.

The roentgenologist reported calcified growths at the distal ends of the first and second phalanges of the middle finger.

At operation, June 2, 1922, five cartilaginous tumors were removed from the sheath of the flexor-tendon of the middle finger (Fig. 21). No bony or fibrous attachment was discovered, and the bodies slipped out easily when the tendon-sheath was cut. These were all about 1.5 cm. in diameter.

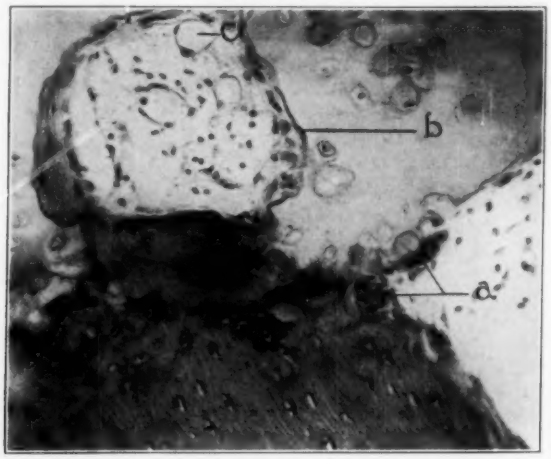

FIG. 22 (Case 15).

Photomicrograph of section of a body shown in Fig. 21; (a) osteoclasts; (b) osteoblasts; (c) capillaries. Note suggestion of ossification by preformation in cartilage.

The bodies slipped from the tendon-sheath and were thought to have been free. However, microscopic examination of sections showed that there was vascular connective tissue on the surface of the bodies. The bulk of the body was made up of lobules of well-preserved hyalin cartilage, but near the vascular connective tissue at the surface there were plates of well-preserved proliferating bone. Figure 22 shows such an area with its blood-vessels, osteoblasts, and osteoclasts.

Case 16 (A 420952). Osteochondromatosis of the right elbow. The patient, a coal miner, thirty years of age, came to the Clinic March 23,

1923, because of pain and limited motion in the right elbow. About 1910, while playing baseball, he had thrown his right elbow "out of joint." The joint had felt weak and was painful when used. No other joints had been involved.

Palpation revealed the crepitus of numerous small hard bodies located in a pouch lateral to the olecranon. The roentgenogram indicated the presence of multiple osteocartilaginous bodies within the right elbow-joint.

FIG. 23 (Case 17).
Osteochondromatosis of the ankle, showing enlargement of the joint.

The very typical appearance practically established the diagnosis even before operation. The roentgenograms made before operation were unfortunately destroyed.

At operation, March 27, 1923, about 125 loose bodies were removed from the joint. Most of the bodies seemed to come from the olecranon fossa; a few were pedunculated and attached, but most of them were free. It was apparent that the entire synovial membrane was not involved in the process.

Two of the bodies were sectioned and found to be composed of fibrous connective tissue surrounding a spherical body of fibrocartilage, which was necrotic and calcified at the center, but well preserved near the surface.

Case 17 (396320). Osteochondromatosis of the right ankle-joint. June 28, 1922, the patient, a farmer, twenty-eight years of age, came to the Clinic because of a swollen right ankle. Four years before, he had bumped the ankle against the wheel of a hayrake and it had pained for two weeks. Ever since then he had noticed an enlargement of the ankle which had gradually increased. The complaint was stiffness rather than pain.

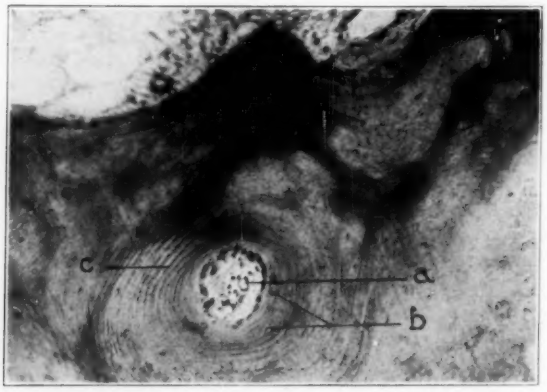

FIG. 24 (Case 17).

Photomicrograph of section of body from the ankle-joint to show reproduction of Haversian canal; (a) central vessel of canal; (b) lacunæ; (c) concentric lamellæ.

Examination revealed a diffuse firm enlargement of the right ankle. It was not tender and there were no signs of inflammation.

The roentgenologist reported multiple calcified bodies in the ankle-joint. January 26, 1923, fourteen osteocartilaginous bodies were removed from the right ankle-joint by two anterior and two posterior incisions. The bodies had so distended the soft tissues of the joint that it was not possible to demonstrate their origin from the synovial membrane.

I am convinced that this is a case of osteochondromatosis, not only because the roentgenogram revealed regular joint-surfaces associated with multiple bodies, but because a cross section of a body showed the typical spherical shell-like structure by which the bodies of osteochondromatosis

often develop. The fibro-osteocartilaginous shell was filled with fat and spurs of bone. There was an abundant blood supply, which accounted for the actively proliferating bone which formed the shell about the center of fat. A fairly perfect Haversian system was found in one region (Fig. 24). The cartilage was very well preserved and in certain areas it showed evidence of rapid proliferation, for the cells approached closely the embryonic type (Fig. 25, a). In other areas the cartilage was more differentiated (Fig. 25, b). In a way the vascular fatty tissues resembled bone marrow (Fig. 25, c). However, there were no marrow-cells, such as normoblasts and myelocytes.

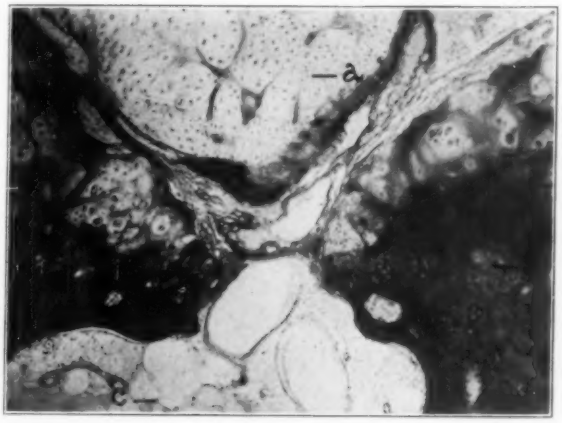

FIG. 25 (Case 17).

Photomicrograph of another area of section shown in Fig 24. Note the various stages of differentiation of cartilage; (a) undifferentiated cartilage (cells are stellate); (b) cartilage relatively well differentiated; (c) fatty tissue resembling bone-marrow.

Case 18 (A 38869). Probable osteochondromatosis of the left knee-joint. The patient, a machinist, twenty-eight years of age, was examined June 14, 1910. Three and one-half years before, he had first noticed a "stinging" in the left knee, which had gradually become more stiff and sore. He had never been compelled to use crutches. At times he had had sudden pain and had not been able to move the leg for a few seconds.

Examination revealed atrophy of the left leg, a swelling about the left knee, and much crepitation. No moving body could be found. At operation, June 16, 1910, about 200 small cartilaginous bodies were re-

moved from the left knee-joint. A part of the dislocated internal semilunar cartilage was excised.

Unfortunately the bodies were not preserved for study and the X-ray plates were destroyed. The large number of bodies and the age of the patient would favor a diagnosis of osteochondromatosis, but in the absence of more proof the case has been classified as probable osteochondromatosis.

Case 19 (A 272634). Probable osteochondromatosis of the right elbow-joint. The patient, a farmer, twenty-six years of age, registered May 29, 1919. Seven years before, he had fallen and injured the right elbow as he

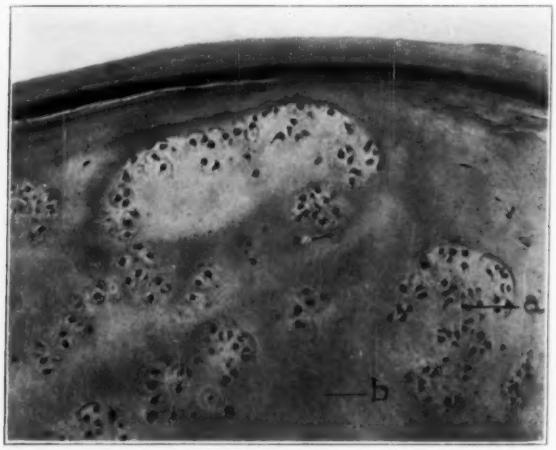

FIG. 26 (Case 3).

Section of a small, pedunculated, cartilaginous body, attached to the synovial membrane. Note structure of cartilage; (a) Clumps of relatively undifferentiated cells; (b) hyaline matrix.

caught himself. Since then the elbow had pained, especially in throwing, and the joint had locked frequently. Extension was limited to 135 degrees and flexion to 50 degrees. Pronation and supination were practically absent. Slight crepitus was present, but there was no pain. There had been slight rheumatic pain in the knees. The roentgenogram revealed multiple loose bodies of the elbow.

December 11, 1920, twelve loose bodies were removed from the joint. Some synovial fringes were also removed. Marked hypertrophic arthritis was present. The pathologist reported osteocartilaginous loose bodies.

PATHOLOGY.

Origin of the Bodies. A matter of interest is the place of origin of the osteochondromas; first, in regard to the layers of the joint-capsule; second, as to which part of the joint-cavity is first involved. However, before reaching any conclusions as to the point of origin, it will be well to review the anatomy of the joint-capsule. The joint-capsule is composed of two main layers: an outer stratum fibrosum and an inner stratum synoviale. The stratum fibrosum is composed of dense connective tissue, while the stratum synoviale is made up of loose connective tissue which contains fat, many blood-vessels, and lymphatics. The stratum synoviale is covered on its inner surface by a smooth layer composed of parallel fibers of connective tissue and round or stellate cells that have large nuclei. In places where there is unusual pressure, these cells with the large nuclei are infrequent; in other areas they are arranged in a single layer, while, especially in the crypts and pockets, this mesothelium may be three or four cells in thickness (Lewis and Stöhr). It is quite natural that considerable interest should have been manifested in the question of the point of origin of the bodies. Laennec, as has been noted above, believed that the origin was in the sub-synovial connective tissue. Brodie believed that the bodies usually had their origin in the synovial membrane itself, but he also said that some specimens in the St. George Museum showed that the point of origin had been outside the synovial membrane in contact with the sub-synovial tissue. Kölliker stated that the bodies develop from the membrane itself and not external to it. Rokitansky held that either the subserous cellular tissue, or the serous tissue itself, might produce the bodies. Eden believed that the synovial membrane was the source, and he claimed to see the stages of transition from synovial cells to cartilage cells in microscopic sections. Kopp found the proliferation just under the mesothelial layer in the synovial membrane.

Findings in material studied in the cases of the present series point to an origin in the stratum synoviale. Figure 20 of Case 14 shows a villus containing small flakes of bone. This villus has nothing to do with the stratum fibrosum, but is composed of the loose connective tissue covered by the mesothelial cells. The finding of bodies external to the synovial membrane, as reported at times in the literature, is not to be doubted. It is possible that some of the outermost cells of the stratum synoviale gave origin to the bodies, and that the bodies failed, perhaps by lack of external pressure, to be forced into the joint-cavity. It is also probable that various writers have different conceptions as to the amount of tissue which con-

stitutes the synovial membrane. When Laennec referred to the subsynovial connective tissue as the place of origin, he possibly had in mind the loose connective tissue or outer part of the stratum synoviale. It is safe to conclude that the stratum synoviale is the structure within which, or in connection with which, the osteochondromas are usually developed. Another point of interest is the determination of the portion of the surface of the synovial membrane which seems most prone to produce the bodies. Hagemann found the formations most numerous in the folds and pockets of the synovial membrane. However, the entire synovial membrane was involved. He thought that this special proliferation in the pockets was explained by the fact that the mesothelial cells are here normally in a layer which is three or four cells in thickness. Case 16 of the present series illustrates the grouping of the bodies in a pocket near the olecranon fossa. Whether the bodies have developed more rapidly in the pockets or have wandered here from lack of space elsewhere is still a question. Müller found in his case that the point of origin was at the juncture of the joint-capsule and the periosteum. Kienböck mentioned this place of origin in addition to the free synovial membrane. Special proliferation in this region has also been noticed by Henderson. Thus, any portion of the synovial membrane may serve as a site for the production of the osteocartilaginous structures, but special proliferation has been noted in the region of the attachment of the joint-capsule to the bones.

It would be interesting to determine whether the bodies start as osteomas or chondromas. Kopp held that they start as osteomas, and not as chondromas, because he found no cartilage about the smaller bodies. Lotsch described the formation of true hyaline cartilage in the synovial villi. In the interior of the hypertrophied villi he found nests of cartilage-cells, which resembled somewhat the appearance of a gland in section. This must be an example of an origin as a chondroma. Material studied in the present series proved that the bodies could start either as osteomas or chondromas. For instance, in Case 3 a small pedunculated cartilaginous body illustrates the origin as a chondroma. Figure 26 indicates the cartilaginous structure. On the other hand, in Case 14 the small flakes of bone found in the hypertrophied villus (Fig. 20) portrays the origin as an osteoma. Hence, it is safe to conclude that these synovial bodies may originate either as osteomas or chondromas.

Body Growth. After the discussion of the origin of these synovial osteochondromas, a consideration of their manner of growth is in order. Descriptions, photographs, and microphotographs have already been presented to illustrate and describe how the bodies enlarge, become pedun-

culated, and then detached to form loose bodies (Case 7, Figs. 5, 6, 7, 8 and 10). The number of years of symptoms mentioned by practically every patient proves that the growth is slow, as does the absence of mitotic figures in microscopic sections. The lack of metastasis and the marked tendency of the process to remain localized are important. In addition, it is important to note that enlargement is by expansion rather than by infiltration.

Kienböck has described several stages in the development of the condition: (1) A small isolated knot in the synovial membrane; (2) several different large knots; (3) diffuse involvement with synovial pouches and bursæ sharing in the formation of bodies; (4) joint-cavity completely filled, the capsule stretched and bone destroyed; (5) bone destroyed deeply, the capsule broken through, and osteocartilaginous masses to be found in the near-by musculature and subcutaneous fat.

The first two stages are not likely to produce enough symptoms to bring a patient to operation. The cases in the present series were all more advanced. Some of them might be placed between stages 2 and 3. Case 3 illustrates very well the degree of involvement described in stage 3, while Case 17, with its distended joint-capsule and destroyed bone, falls definitely into stage 4. Stage 5, with the bodies in the muscles and subcutaneous fat, is more advanced than the condition present in any case in the series. The involvement of the knee-joint reported by Lexer belongs in this class.

In the study of the growth of the cartilage and the bone, attention will first be directed to the conditions present and the changes that take place before detachment. The cartilage may be purely hyaline, fibrous, calcified, or a combination of these forms. The cartilage in the attached bodies was for the most part found to be well preserved. However, in the interior of some of the larger cartilaginous masses there were evidences of retrogressive changes, such as calcification, enlargement of the spaces in the matrix, pyknosis, and karyolysis.

Another point of interest in studying the growth of the cartilage was the lobulated form which was so noticeable. At times these lobules were found to be separated by fibrous connective tissue, and, at others, the septa were of a hyaline structure. There appear to be two possibilities that may explain the lobulated appearance: either the growing cartilage sends off buds, or, more likely, the separate lobules have developed from isolated knots of cells in the synovial membrane and have fused into a single mass by stretching and crowding out the connective tissue lying between the lobules.

In the study of bone growth in the attached bodies, there was evi-

dence to indicate that bone was developed directly from the connective tissue by the membrane method and also by preformation in cartilage. The bone shown in Fig. 13, Case 7, appears to have been developed by the membrane method, for there is no cartilage near the thin plates of bone. On the other hand, the bone in Fig. 22, Case 15, was probably preformed in cartilage, for the thin layer of bone lines a vascular cavity in the cartilage, producing a picture very similar to that seen in a section of a developing bone in the embryo. In relation to the nutrition, these observations were made: that in each case in which there was vital proliferating bone, there was also a blood-supply; but that, when the blood supply had been cut off, there were retrogressive tissue changes (Case 7, Figs. 11 and 13) (Case 15, Fig. 22) (Case 17, Fig. 24). Also osteoblasts were present only where there was a blood supply.

Next in order in the study of the growth of the bone comes a consideration of the structures noticed. The most typical form was the spherical shell of bone surrounded by cartilage and fibrous tissue and filled with vascular fat and spurs of bone. Such a structure was described in the case reported by Hahn. In the series reported here, this structure is very typically shown in the pedunculated body in Case 7 (Fig. 8), in a large body in Case 14, and also in a large body in Case 17. The resemblance to the structure of normal skeletal tissue is very striking indeed. There is the shell of bone whose cross section corresponds in shape to the ring of bone seen by cross section of the shaft of a normal long bone. In the loose body the shell is surrounded by cartilage, which may be compared to the cartilage which covers the articular ends of the bones of the skeleton and it is filled with vascular fat which represents the marrow of the long bones. Though this fat contains spurs of bone which is often lined by osteoblasts and osteoclasts, there are no myelocytes, normoblasts, or other blood-forming cells which distinguish normal marrow. Varying stages of differentiation were reached in different cases, and forms more disordered than those just described were found. For instance, in Case 15 osseous tissue was found in small amounts at the periphery of the cartilaginous mass. Osteoclasts were not always found when osteoblasts were present, and this is not strange for osteoclasts are associated with resorption of bone.

Attention will next be directed to the conditions present and the changes that take place in the bodies after detachment. The fate of the cartilage in the detached body is the first problem presented. Undoubtedly, there are retrogressive changes that take place, especially at the center and deep within the masses. Especially noticeable in the detached bodies

are the concentric laminations (Fig. 10). Fisher has pointed out that these laminations are determined by zones of calcification, and this finding has been substantiated in the present study. Marsh stated that the bodies most likely ceased to enlarge after detachment. Dick also raised the question as to whether the bodies grow after detachment, and he hoped that future observations might solve the problem. Carothers said that the question had been discussed in the literature, but not satisfactorily settled. Kappis concluded that cartilage did proliferate after detachment. Strangeways claimed to have observed very definite evidences of cartilaginous proliferation in loose bodies. Fisher thought it clear that bodies continue to grow while free within the cavity of a joint. To substantiate his view, he speaks of the case reported by Marsh in which the small bodies had traces of pedicles, while the larger ones had not. In the examination of the pathologic material, evidence favoring the possibility of cartilaginous growth after detachment was not conclusive. In the first place the finding of well-preserved cartilage near the surface of a loose body did not prove that the cartilage was proliferating; moreover, numerous free bodies were found with marked retrogressive tissue changes even at the surface. On the other hand it was the rule that the small bodies showed evidences of pedicles, or were definitely attached to the synovial membrane, while the larger bodies were generally detached.

If these bodies do grow after detachment, the problem of the source of nutrition is immediately raised. There are grounds for doubt as to whether the synovial fluid furnishes the total supply of nutriment for the articular cartilages; but in the case of the detached bodies it must be the only source, unless it is possible that there might be a transfer of substances by direct contact of the cartilage-cells to the vascular synovial membrane. In accounting for the enlargement of detached bodies, the cartilage has been credited with the growth, because, whenever a detached body was sectioned, the osseous tissue was found to be necrotic (Case 7, Figs. 10 and 11). The same observation was made by Fisher, and Kappis thought it very doubtful whether real bone formation could take place in a free body. He observed that when a free body contained bone, the bone was necrotic.

Hence, it is possibly true, but not conclusively proven, that cartilage may proliferate when free in the synovial fluid. On the other hand, it is certain that growing bone demands a blood-supply.

After detachment, it is possible that the bodies may form a new attachment to the synovial membrane. Barth learned by animal experiments that artificially detached pieces of the articular surface always

became attached to the synovial membrane. Large bodies in Cases 15 and 18 were found to have a vascular connection, and it is possible, but not necessary, that this came about by a reattachment. The formation of a small quantity of bone on the outside rather than in the center of the large cartilaginous mass in Case 15 (Fig. 22) suggests that the bone formation was an aftermath and perhaps came by reattachment. However, this is only conjecture.

ETIOLOGY.

Closely linked with the pathology of synovial osteochondromatosis is the question of etiology. Four theories will be considered: the infectious, the traumatic, the embryologic, and the neoplastic.

The infectious theory. Attention will first be directed to the views expressed in the literature. Brodie said that in a majority of the instances there was no evidence of previous inflammation. Bibergeil and Eden excluded arthritis deformans when considering the etiology. Reichel spoke of a contagious agent that involved the whole joint-cavity. However, he was undoubtedly not speaking of a contagious agent of an infectious nature, for in the report of his case he specifically mentioned the absence of an inflammatory process. Carothers thought that infection was the most obvious etiologic factor in his case. Bolton, Halstead, Whitelocke, and Kappis have discussed the formation of osteochondromas in chronic arthritis and synovitis, but it is doubtful whether they were referring to true osteochondromatosis. Fisher quite specifically divided synovial chondromas into the type found in osteoarthritis and the type unassociated with osteoarthritis. However, he stated that the two types were identical as to gross and microscopic appearances. This division seems most logical.

The clinical histories, physical findings, and pathologic examinations of the cases in the present series were closely studied to determine, if possible, an infectious factor in the etiology. The lack of redness and heat in the reports of physical findings, the absence of arthritic changes in multiple joints, which is so common in infectious arthritis, and the very characteristic preservation of the normal articular surface in so many cases, as was evidenced especially by the roentgenograms, argues against an infectious origin. In most cases in which roughness of the articular surfaces was encountered, it was only of a degree that could have been caused by the intra-articular bodies. The perivascular lymphocytic infiltration in Case 7, Fig. 14, might be considered a support for the infectious theory. How-

ever, this may be explained as an evidence of chronic inflammation due to trauma, and not necessarily infectious in nature.

It is safe to conclude that infection does not play an indispensable or important part in the etiology of synovial osteochondromatosis.

The traumatic theory. The views expressed by Humphry, Davis, Carothers, Henderson, and Fisher with regard to the relationship of trauma to the etiology have already been given. In the cases reported by Reichel, Riedel, and Bibergeil no trauma was known of. In general, the German observers have given little weight to the consideration of trauma as a cause. There has been a different trend of thought in the English and American literature. Marsh wrote of the enlarged synovial villi, which became converted into cartilage in the course of synovitis of long standing, which was provoked by injury (or due to osteoarthritis). Whitelocke and Heineck (also Rehn and Eden, among the German authors) made similar statements. Trauma figured in the history of two cases reported by Kopp. In twelve cases collected by Lotsch, trauma was mentioned by several of the patients, but Lotsch did not consider it a causal factor. Carothers found evidence of hemorrhage in some of the synovial villi, and accounted for the pathologic calcification on the grounds of traumatic hemorrhage.

Trauma figured quite definitely in the histories of eight of nineteen cases in the present series. In Case 1 there had been a month's disability, following injury to the joint five years before the first symptoms of the trouble for which the patient came for treatment. In Case 2 the joint had been injured ten years before the date of examination, and this injury was so severe that swelling had persisted for six months. In Case 8 an injury to the joint, twenty-five years before the first symptoms, had disabled the patient for three weeks. In Case 11 the forearm had been injured by a pitch-fork and the elbow traumatized by striking the joint against a sharp steel projection which had caused intense pain and residual soreness. In Case 14, nearly twelve years before the examination, the patient had fallen on the elbow, and there had been pain for a week, with swelling which had persisted more or less ever since. In Case 15, in which five osteocartilaginous bodies were removed from the flexor tendon-sheath of a finger, trauma was very definite. Six months before symptoms began, this finger had been dislocated backwards by a baseball. It may be said in several of the instances cited that the trauma only produced alarming symptoms, because the pathology was already present; but in the last instance this objection can hardly hold.

From the opinions expressed in the literature, from the preponderance

of males in the series of cases, and from the important place given trauma in the histories of cases reported, it is reasonable to place considerable importance on the rôle that trauma plays in the etiology of synovial osteochondromatosis.

Embryologic theory. This theory, advanced by Lexer, has already been stated. Whitelocke, Lotsch, Henderson, and Fisher also point out the fact that all the tissues involved are developed by differentiation of the same mesenchymal tissue. In the embryo, before a joint is formed, the cartilage which represents the future bones is separated by undifferentiated mesothelium. Some of these cells undergo mucoid degeneration and form a joint-cavity; other cells become spindle-shaped and form articular cartilage, while the cells at the sides of the cavity form synovial membrane. The bursal sacs, which are often connected with the joints, undoubtedly are closely related developmentally to the joints. As we do not know why one mesenchymal cell forms a mucoid fluid while another forms cartilage and another synovial membrane, it is not possible for us to determine exactly what abnormal forces could cause cartilage to develop in the synovial membrane.

It is sufficient here to say that the theory is quite logical; that the consideration of the tissues in their developmental stages helps in the approach to the problem; and that this theory does not contradict any of the other logical theories advanced. Indeed, the discussion of the problem from a developmental standpoint leads directly to the consideration of this process as one of the tumors.

Neoplastic theory. In the review of the literature, there were presented the views of Brodie, Müller, Henderson, Fisher, Troell, and others with regard to the possible relationship that synovial osteochondromatosis bears to tumors in general. In casting about, reports of various other benign intra-articular tumors of the synovial membrane have been discovered. Reichel drew attention to the very generally known synovial fatty tumor often referred to as the lipoma arborescens. Several cases of synovial fibromas are on record. Robson reported a case in which the patient sustained a contusion of the knee two years before his examination. He found a swelling and thought that he would find a foreign body in the joint. Operation disclosed three tumors attached to the synovial membrane by broad bases. By microscopic examination they were found to be fibromas. They were compared in size to a hazel nut, a walnut, and a hen's egg. Von Brunn[26] (1907) examined an eighteen-year-old girl, who had had a swelling of the knee for two years. Operation revealed a fibroma of the lateral meniscus. In the article by Bryant, published in 1902,

mention was made of a fibroma which was found attached to the synovial membrane by a pedicle. Duda[26], in 1894, reported his findings in the case of an eighteen-year-old male who complained of pain and stiffness which had been present in the knee for two weeks. At operation a walnut-sized tumor was found hanging to the capsule. It was found by microscopic examination to be an angioma. In addition to the benign tumors, mentioned above, there are numerous reports of malignant involvement of the synovial membrane. Brann in 1869, Steudener[26] in 1869, Garré in 1891, and Langemak in 1904 each report a case in which a sarcoma had involved one of the bones near the knee-joint and the capsule or synovial membrane of the knee-joint. The primary growth was probably in the bone in each case. However, this was not certain. Henderson, in 1916, reported the case of synovial osteochondromatosis associated with malignancy of the distal end of the femur. This is reported in detail as Case 1 of the present series. There are several points of special interest about this case: the osteochondromatosis was associated with malignancy of a near-by bone; the process by which the synovial osteochondromas were formed was probably not a manifestation of a malignant tumor, for the osteochondromatosis did not recur; the association of these two tumors pointed to a developmental disturbance. These examples have been given to illustrate the range of tumors of the synovial membrane from the very benign to the very malignant. The malignant growths mentioned have little in common with synovial osteochondromas, but all of these tumors are developed from the same region of mesenchymal tissue in the embryo, and the consideration of the whole field helps in the placing of the synovial osteochondromas.

Another fact which supports the neoplastic theory is the relationship between trauma and the development of synovial osteochondromatosis. Admitting the limitations of *post hoc, ergo propter hoc* argument, cognizance must still be taken of the fact that very often the occurrence of definite trauma before the appearance of a neoplasm can hardly be explained as an unrelated incident.

The various stages of differentiation found in the microscopic examination of the pathologic material strongly support the neoplastic theory. Fig. 25 (a) illustrates undifferentiated cartilage, whose cells approach the mesenchymal cell in appearance. Fig. 26 (Case 3) shows further differentiation, but even these cells are intermediate in type, for they have been dividing so rapidly that the cells are massed in clumps with cartilaginous matrix only between the large clumps. On the other hand, Fig. 12 (Case 7) illustrates well-differentiated cartilage. It is a well-recognized fact that

there is a tendency in all neoplasms, except the very malignant undifferentiated growths, to attempt to reproduce in an imperfect way some normal structure. Support for the neoplastic theory may be seen in the repeated attempt found in synovial osteochondromatosis to reproduce the normal structure of bone by forming the shell of bone outside of which is the cartilage, and in the center of which is the fatty, vascular tissue representing the marrow (Fig. 8). Another indication of this imperfect attempt to reproduce the structure of the normal bone is found in the Haversian canal, seen in Fig. 24 (Case 17).

Before reaching any conclusion, it would be well to define the term "neoplasm." According to Ziegler, a neoplasm is "a new formation of tissue apparently arising and growing independently, having an atypical structure, inserted uselessly into the organism, possessing no function of service to the body and showing no typical termination of growth." A benign neoplasm is characterized [54] by an expansive rather than an infiltrating growth, by slow growth and the absence of mitotic figures, by being encapsulated, by never metastasizing, by not recurring after complete removal, by being composed of well differentiated cells, and by not inducing cachexia. Synovial osteochondromatosis may certainly be classified as a neoplasm, and so far as we know at present it is of a benign nature.

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PATELLAPEXY—AN OPERATION FOR THE RELIEF OF PARALYSIS OF THE KNEE.

BY DR. WALTER G. STERN, M.D., F.A.C.S.

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For the substitution of the paralyzed quadriceps muscle three methods have been used:

First: Resection of the joint, with bony ankylosis, either in the extended or slightly flexed position. This, of course, is successful in giving firm and steady limbs, but when done before the age of puberty interferes so markedly with the growth of the limb that the operation has deservedly fallen into disuse.

Second: Transplantation of the hamstrings into the patella. The prime requisites for this operation are an absolutely straight knee and strong hamstrings, a combination not always found. Some of these operations have yielded most brilliant results, but on the whole I believe our failures have been as numerous as our successes.

Third: In order to reproduce the conditions found in children with paralytic knees who walk readily and surely when the knee is held hyperextended, the late Dr. G. G. Davis, of Philadelphia, proposed the fracturing of the femur just above the knee and the fixation of the lower fragment in such a position that weight-bearing causes the knee joint to tend toward hyper-extension rather than flexion and so stabilize the weight-bearing joint.

Having noted the effects of tendon fixation for the relief of paralytic conditions in other joints (chiefly in the foot) it was apparent to the writer that failure was usually due to the compulsory use of long, weak tendons working on the end of a long lever (as for instance in the correction of varus, valgus, or drop foot), and the best and surest results were obtained where strong short tendons working on a short lever (as in the fixation of the Tendon Achilles to the tibia for calcaneus) were employed.

It, therefore, occurred to the writer, in the summer of 1922, to fix the patella to the lower end of the femur in chosen cases of infantile paralysis in order to stabilize the knee joint and keep the knee from flexing while bearing weight. The ideal conditions for permanent tendon fixation—short, sufficiently strong tendons, not running through yielding, soft tissues and working on a short lever—would be met. For our first operations

only cases of complete quadriceps and hamstring paralysis of the knee were operated upon, so that no question of the propriety of a tendon transplantation could be raised. In all, eight cases are ready to be reported upon.

The operation is a simple one. A vertical incision over the patella about four inches in length allows the anterior surface of the patella to be completely denuded of cartilage down to the center of ossification of the patella. These undesirable portions of patella need not be excised, but can, except for a small triangular piece in the center, be included in the lateral flaps. The quadriceps tendon, patella and its tendon are separated from the lateral fascia and the capsule of the knee joint by two vertical incisions, one on either side of the patella, each about four inches in length. The patella is then retracted to one side and a bed prepared for it in the femur. The patella is then turned around 180 degrees on its long (vertical) axis and nailed or screwed firmly to the femur. The lateral tissues are sewn over as tightly as possible to what was formerly the posterior surface of the patella, the skin closed and a cast put on with the knee in complete extension. The rotation of the patella permits an easier and more complete denudation of non osseous tissue of the patella by working on its anterior surface—and allows of the taking up of a certain amount of "slack" in the quadriceps tendon system and also mechanically tends to keep the denuded surfaces together by the tendency of the patella to rotate back again, while if the posterior surface of the patella were used the patella would have the tendency to ride away from the surface of the femur.

So far, I have not been able to stabilize any joint in complete extension; five to ten degrees of flexion motion has remained. This will not be a bad fault as it allows the patient to walk easily and to sit down in comfort. The nails have been well borne, but I removed one four months later under local anesthesia to more clearly radiograph the ankylosis of the patella to the femur. The cast is left on for three months, after which I have allowed the patients to walk for another three months in caliper splints in order to guarantee a complete fixation. I do not anticipate that the tendon of the patella will stretch too much to undo the stability of the joint, although several years must elapse before final judgment can be passed in this point, and I anticipate the patella and its tendon will grow proportionately to the growth of the epiphyses. If the tendon of the patella does not grow it will lead to complete extension of the knee—not an undesirable condition. All my patients can hold the leg extended on the thigh, walk without braces to support the knee, and can sit down in comfort.

The operation is indicated in all cases of paralysis of the quadriceps which cannot walk without braces, in which the hamstrings are also paralyzed or weak, or, if the hamstrings are good, in which the knee cannot be fully extended, and in cases in which transplantation of the hamstrings has either been unsuccessful or undesirable.

DISCUSSION ON PAPER OF DR. STERN.

DR. A. H. CILLEY, New York City: I do not see why this should not be a perfectly good expedient while you are waiting for growth before you excise the knee-joint. I do not see how it would be of value in later life because of the weight and because we know that paralyzed muscles stretch.

DR. W. G. STERN, Cleveland, O. (closing the discussion): I think Dr. Lovett's criticism is sound but the operation of Patellapexy in no way adds to or causes any genu recurvation. It may even cure (symptomatically) this condition, by so stabilizing the knee that walking with a slightly flexed knee is possible and then by raising the heel this position is forced upon the patient.

EVULSION OF THE POSTERIOR CRUCIAL LIGAMENT OF THE KNEE JOINT.

BY T. K. RICHARDS, A.B., M.D., BOSTON, MASSACHUSETTS.

THE following case seems to be worth recording because of the unusualness of the injury and because conservative treatment produced a perfect functioning joint.

FIG. 1.

A young man of twenty, playing varsity football, turned suddenly to the right in an attempt to tackle the runner. The cleats of his right shoe held so firmly in the turf that the full weight (208 pounds) of his body in turning to the right caused the upper leg to be twisted on his lower leg at the knee joint. "Something snapped" and the man collapsed. (Note

there was no external violence. He was not run into nor did he strike anybody). The knee was locked in about sixty degrees of flexion. He was suffering extreme pain but could not localize it. No cartilage could be felt out of place and no points of tenderness were elicited. There was no crepitus.

Because of the manner in which the injury had occurred it was thought that the locking must be due to a dislocated internal semilunar cartilage.

FIG. 2.

Accordingly the joint was put through the customary manipulations, that is, the joint was unlocked so that the leg could be straightened. The acute pain, however, persisted—which is not the usual happening with dislocated cartilages after they have been reduced. The knee was held in about thirty degrees of flexion and could be straightened only with excruciating pain.

A radiogram (Fig. 1) showed an irregular rectangular hole on the internal superior aspect of the intra-condylar notch, at the site of the attach-

ment of the posterior crucial ligament. The space between the femur and tibia was greater than normal.

The knee was examined again and it was found that the tibia could be rotated inward and backward on the femur. The knee was still somewhat flexed.

A diagnosis of an evulsion of the posterior crucial ligament from its femoral attachment, with the breaking off of a small piece of the femur, was made. This fragment of bone had apparently locked the joint.

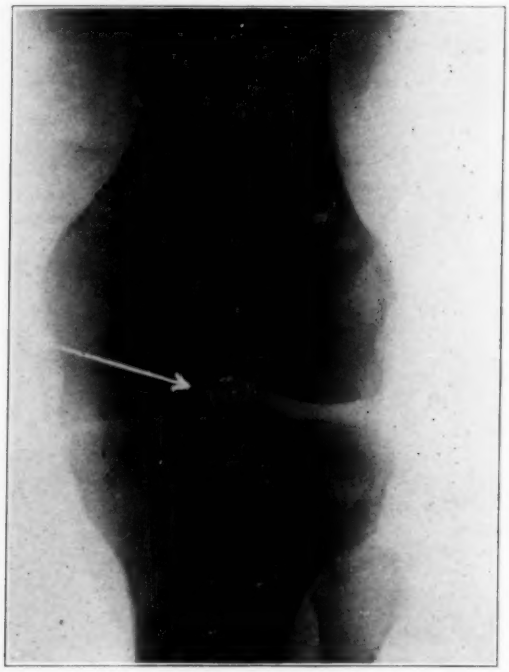

FIG. 3.

Inasmuch as the intra-condylar notch is the only portion of the femur in the knee joint that is covered with periosteum and since it was unlikely that the ligament had been torn in two places it was decided to try to fully extend the knee by gradually forcing it down on a ham splint with elastic bandages. The idea was to try to force the fragment of bone attached to the ligament into the intra-condylar notch and trust that there would be sufficient reparative power to make the fragment stick.

After thirty-six hours of bandaging, the knee was found to be straight. A high plaster of Paris cast was applied with the knee hyper-extended. A radiogram (Fig. 2) at this time still showed the deformity in the side of the intra-condylar notch but the space between the femur and tibia was now normal.

The cast was removed at the end of three weeks. At this time the patient was able to move his knee back and forth through an arc of thirty degrees. The abnormal mobility had disappeared. He was started on massage and active motion. At the end of four weeks he was walking without crutches, and at the end of two months from the injury the patient was walking without a limp and without artificial support. A radiogram (Fig. 3) at this time failed to reveal any deformity in the intra-condylar notch.

The patient states that the only difficulty he has with his knee now is that after sitting for some time the joint feels cramped and gets a little stiff. This disappears as soon as the leg is moved.

ADULT TORTICOLLIS

REPORT OF A CASE.

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WHILE surgical treatment of torticollis in children generally gives good results when properly done, in adult torticollis the result is often disappointing, inasmuch as the operation is productive of no results at all in some cases, while in others it may actually aggravate the condition. The reason for this is obvious. In torticollis of children the obstruction is in

FIG. 1.
Before the operation. Note height of right shoulder.

the contractures of the muscles and fascial bands. The thorough division of the contractures and the holding of the head in the over-corrected position for some time will invariably yield even excellent results. In adult torticollis, however, the obstruction is not only limited to the soft parts, but may also co-exist in the vertebræ. In the latter instance, if an actual

bony ankylosis exists, the division of the contractures would not correct the deformity. Moreover, in some cases of adult torticollis the operation only aggravates the asymmetry of the face, and the patient looks even more deformed than before the operation. Sound judgment, therefore, as to which cases are to be operated upon and which are to be left alone is the prima facie of success in adult torticollis. A successful operation for a severe type of adult torticollis is therefore worthy of record.

FIG. 2
After operation.

Patient aged 29. Severe congenital torticollis. His head and shoulder were drawn together to such an extent as to make these two appear as one continuous mass, and the neck on the affected side was entirely obliterated. When the patient walked with a person on either side of him, he cou d see only the one who was on the left side. Due to excessive brooding, the patient had developed a psychic complex. He was sure that people were staring at him and they were whispering the word "cripple" as they passed. Gradually his determination grew not to go through

with life. When he came to me he had already been rejected by a number of surgeons as a poor surgical risk on the ground that he was too old and that the "bones had already set."

Operation. The skin was pulled up over the clavicle in order to place the scar as low down as possible. A well-developed contracted platysma was encountered, which was divided. Both heads of the sterno-cleido mastoid were also divided. The trapezius and the scaleni were also cut. The main obstacle to correction, however, consisted of bands which derived their origin from the deep and superficial cervical fascia and ran in all directions, holding the head and shoulder in the deformed position. These were divided. Finally the vertebræ were gently but firmly manipulated and the head put up in an over-corrected position in a plaster of Paris cast.

Result. The shoulder dropped to the same level as the opposite one. The head became straight and the neck spacing about normal, so that the patient, who never before was able to wear a collar, not only wears one now, but makes it a point to wear the highest collar he is able to get. Because the cervical vertebræ have straightened, he is two inches taller than before the operation. His facial asymmetry has improved in the last three years.

In order to insure good results in the operative treatment of adult torticollis it is necessary to ascertain that no true bony ankylosis exists between the cervical vertebræ and that the new position will not aggravate the asymmetry. Also one must divide and thoroughly stretch the contracted muscles and fascia and for some time hold the head in an over-corrected position, or until the tendency for a recurrence has been overcome.

A "BASEBALL FINGER" CURED BY OPERATION.

BY RICHMOND STEPHENS, M.D., NEW YORK CITY.

IT is not unusual to see a so-called "baseball finger" and in my experience the majority of them have remained uncorrected, with permanent disability or at least inconvenience. The cases with bony injury are naturally less favorable for treatment, but in some of the cases, as the one here reported, there is only the tendon injury, and these I feel certain can be cured. The following case was seen six weeks after injury and after three weeks of conservative treatment. The condition was very annoying to the patient and he was extremely anxious to have it corrected.

H. L., clerk, 17 years old. Patient was hit by a baseball on the tip of the right middle finger about June 1, 1923. The injury was quite painful and the finger became "crooked." For several days there was swelling and tenderness and the deformity persisted. He was untreated for three weeks, then had a splint applied for the next three weeks, and in his opinion there was no improvement.

On July 11, 1923, the patient was admitted to the Hospital for Ruptured and Crippled, Second Division, and at this time presented flexion deformity at the distal inter-phalangeal joint of the right middle finger. The distal phalanx could be fully extended passively but not actively. A roentgenogram did not show any pathologic condition. The diagnosis was made of a separation of the extensor tendon at its insertion into the last phalanx.

Operation, July 12, 1923. Through a longitudinal incision over the dorsum of the right middle finger, extending from just back of the nail bed to the middle of the second phalanx, the end of the extensor tendon was found free and separated about one-quarter of an inch from its normal point of insertion on the distal phalanx. The tendon was pulled down to the point of the insertion and fixed with fine kangaroo sutures. The skin was closed with plain catgut sutures and an anterior splint was applied. The patient was allowed to go home on the fourth day. The wound healed by primary union and recovery was uneventful. The splint was worn for about four weeks and then active motion was started.

Since that time the finger has appeared to be normal, there is complete active extension of the terminal phalanx with as much power as before, and the patient is entirely satisfied with the result.

Because of the frequency of this injury, the simplicity of the operative cure in cases where conservative treatment fails, and the perfect result obtained in this first case, I wish to recommend the operation to those who may not have already used it.

NOTES ON THE TREATMENT OF CERTAIN FRACTURES OF THE FEMUR.

BY WILLIAM H. BYFORD, M.D.

Junior Surgeon to St. Luke's Hospital, Chicago, Illinois.

FRACTURES through the lower third of the femur are at best difficult of reduction and fixation. The heavy muscles of the thigh pull the lower fragment proximally and those of the calf displace the upper end posteriorly. In the treatment of these fractures it is necessary to overcome the action of both these sets of muscles. This is accomplished by extension followed by fixation in a cast or splints, or by a plate. The extension is usually applied with a double inclined plane splint. If, however, the fracture is close to the knee joint, difficulty is experienced in elevating the lower fragment without resorting to an open operation. To eliminate this procedure the following method was used.

In Case 1 calipers were applied to the condyles of the femur, and the thigh and leg placed on a double inclined plane splint with 35 pounds extension applied to the calipers. At the end of a week the deformity showed no improvement. The splint was then removed and the extension changed so that the thigh was held perpendicularly to the bed. The leg then by its own weight assumed a position of acute flexion on the thigh with the foot extended. The extension of the foot relaxed the tension on the Achilles tendon and the muscles of the calf. The extreme flexion of the knee relaxed the gastrocnemius, which held the lower fragment down. The flexion of the lower fragment of the femur being limited by the capsule and ligaments of the knee joint, and with the calipers acting as a fulcrum, the foot and leg being the heavier and longer arm, applied positive upward pressure to the lower fragment. Reduction was accomplished in three days of this position.

In Case 2 the fracture was nearly three weeks old when first seen. All operative measures were refused and so an attempt was made to duplicate the results obtained in Case 1 without the use of calipers. The results were not as good as in the first case, but this I believe was due to the age of the fracture rather than to any fault in the method. To take the place of the calipers a cast was put on the leg extending from the ankle to

FIG. 1.
Case 1, showing original fracture with Carrel tubes in place.

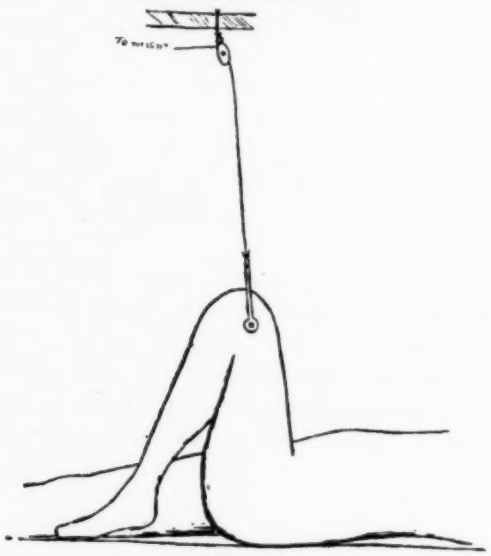

FIG. 2.
Case 1. Method of applying traction.

FIG. 3.
Case 2, showing original fracture.

FIG. 4.
Case 2, showing reduction effected.

a point about two inches below the knee. Extending along the anterior surface of this cast was an extension made of fiber board wound with plaster. This was about six inches long. Thirty-five pounds extension was then applied to this projection of the cast. The weight overcame the contraction of the thigh muscles, and at the same time, the knee joint acting

FIG. 5.
Case 5. Method of applying traction.

FIG. 6.
Case 2. Cast of leg.

as the fulcrum, depressed the foot and swung the lower fragment of the femur towards its normal position. This position was retained for ten days, at which time over-correction was seen to be present, showing the efficacy of this method. This over-correction was remedied at the time the permanent cast was put on. The end result was a slight over-riding of the upper fragment, but good alignment and no shortening.

FIG. 7.
Showing reduction effected.

FIG. 8.
Fracture before operation.

FIG. 9.
Fracture after operation.

The method outlined above is not expected to replace open reduction, but many instances will be found in which it is useful, particularly when open reduction is refused or not indicated.

Case 3 I am reporting through the kindness of Dr. A. E. Halstead, on whose service the patient entered the hospital. Open operation was resorted to first, but following the application of a cast the fragments were seen to have slipped and to be in but little better position than before operation. Pneumonia developing on the second day after operation prevented a second attempt and the application of a plate or other method of fixation. Accordingly the leg was put up in the position described earlier in this article with the result shown in the accompanying photograph.

FIG. 10.
Final reduction with traction described.

LATE ULNAR NERVE PALSY FOLLOWING ELBOW FRACTURE IN THE ADULT.

BY WALTER M. BRICKNER, M.D., F.A.C.S.

Attending Surgeon, Hospital for Joint Diseases, and Broad Street Hospital; Associate Surgeon, Mount Sinai Hospital, New York.

ALTHOUGH first described by Panas[1] over forty years ago, there are probably less than one hundred recorded instances of a distal ulnar neuritis or paralysis appearing from one year to even forty years after an injury to the corresponding elbow. The phenomenon is, however, clearly established by observations that have appeared in the French, German, Italian, British, and American literature.

Mouchet[2] described the affection as resulting from a fracture of the external condyle of the humerus, with considerable displacement, occurring in childhood, and gradually producing a cubitus valgus, distortion of the relationship of the olecranon process and olecranon fossa, flattening of the ulnar groove, and exposure of the nerve to tension and to repeated irritation. Subsequent writers have confirmed Mouchet's observation by their own case reports; and fracture of the external condyle of the humerus in childhood with resulting cubitus valgus has become recognized as the typical injury which three, four, five, or twenty or thirty years later, may be followed by the appearance of an ulnar neuritis or paralysis. Thus of the four American contributions to this subject: Murphy's[3], the first case, was of this character; Hunt[4], who published the first description of the phenomenon in this country, reports three cases, all of this type; and in the latest article that has appeared, Miller[5], who records seven cases observed by him, says:

1. The primary cause in practically all cases of late ulnar nerve palsy is a fracture at the elbow in childhood, usually between the third and fifth year. 2. Although occasionally the site of injury may be at the internal condyle or in the supracondylar region, in the vast majority of cases the line of fracture begins laterally just below the epicondyle and passes obliquely downward and inward into the joint, causing a complete separation of the external condyle (capitellum). 3. The broken capitellum is displaced laterally and forward, and the fractured surface is twisted outward, is not accurately reduced by manipulation, and as a

result non-union of the condyle occurs. The growth of the humerus on its lateral side is thus interfered with to such an extent that a cubitus valgus gradually develops. 4. As the deformity increases the olecranon process becomes impinged against the medial condyle, the ulnar groove becomes but a shallow depression and the nerve itself displaced from its bed, where it becomes subjected not only to stretching when the arm is flexed and extended but to repeated slight trauma, which in time leads to partial or complete ulnar palsy. 5. This paralysis may begin as early as three years or as late as forty years after the injury, but in the majority (40%) of cases it is noticed between the twentieth and thirtieth year after the fracture occurs.

This description, based on a study of previously reported, as well as of Miller's own cases, and corresponding thus with the observations of Mouchet, Hunt, Peltesohn[6] and others, may be accepted as a statement of the pathogenesis of this lesion as it usually develops.

A casuistic analysis of the literature shows, however, that the phenomenon of late ulnar nerve palsy is not always the result only of elbow fractures in childhood nor, indeed, only of fractures. Thus, of the four cases reported by Panas one was an instance of ulnar paralysis appearing at the age of 40 in a man who had fractured his elbow at the age of 28. Of Bowlby's[7] two cases, in one the injury occurred in infancy, but in the other it occurred at the age of 28; in both, the ulnar paresis appeared at about the age of 40. Of the twenty-two cases observed at the Mayo Clinic, as tabulated by Shelden[8], seventeen could be traced to elbow fractures in childhood. Of the five others, all males, the first, aged 42, had had no injury but there was a bony outgrowth; the second, aged 39, had had a fracture at the age of 38; the third, aged 62, had had "an indefinite injury at 52. Hypertrophic arthritis;" the fourth, aged 40, had an "old destructive arthritis" with nerve symptoms for one year; the fifth, aged 61, with bilateral ulnar neuritis, had dislocated his left elbow at the age of 26 and his right elbow at the age of 28. Possibly also in some of the other cases in the literature in which the age at injury is not stated it occurred in adult life.

Mrs. E. M., aged 52, referred to me by Dr. B. S. Oppenheimer on September 29, 1914, had that day fallen on her right elbow, which was much swollen. The injuries she had sustained, as showed by roentgenograms, were: (a) fracture (small fragment) of the olecranon process, not into the joint, with separation of about 1 cm.; (b) infraction (fissure) of the (coronoid) articular surface of the ulna; (c) fracture of the edge of the internal epicondyle of the humerus; (d) infraction (fissure) of the external epicondyle.

In spite of early mobilization, limitation of joint function developed:

1914, October 10—Arm in sling without dressing. Movements encouraged and light massage instituted.

November 4—Still swelling and infiltration in olecranon region. Flexion and extension much limited. Active massage.

November 18—Flexion possible to 85°, extension to 135°.

November 25—After four diathermic treatments; swelling less but function not improved. Pronation and supination unimpaired.

December 16—Joint adhesions broken under N_2O. Full flexion and extension when possible under narcosis. This, however, could not be maintained and on

December 28—Adhesions again broken and elbow splinted in extension.

1915, January 26—Patient can extend to 140°, and flex to 60°.

March 31—Function improved. With arm on table can extend until mid-wrist is six inches from table, and can flex until wrist is eight inches from tip of acromion process. This was the range of motion also on May 1st, seven months after the injury, when the patient was discharged. She had then actively resumed her duties, which involved much handling of books and pamphlets and some typewriting.

On January 17, 1916, Mrs. M. reported to me again with atrophy of the thumb adductor, slight atrophy of the interossei, and paresthesia of the fourth and fifth fingers of the right hand. She was able, however, to contract the adductor pollicis and the interossei, and there were no objective disturbances of ulnar sensation, epicritic or protopathic, except very slight loss of light touch sense. There was no pain. The response of the affected hand muscles to electric stimulation was not as prompt as normal but K.C.C.>A.C.C. No callus felt about the ulnar nerve at the elbow, but the nerve was palpably thickened and the bone groove appeared shallow. The patient stated that the paresthesia had begun some months previously, that she first noted weakness in the hand about three months previously and atrophy about Christmas.

January 29—Examined by Dr. J. Ramsay Hunt, who found slight R. D. and agreed that the ulnar nerve should be liberated at the elbow and moved to a fresh bed. This operation was unavoidably delayed a few weeks, during which the lesion definitely progressed and the electric responses diminished.

March 11—Operation. Brickner. (Mount Sinai Hospital, private pavilion):

Midline incision over extensor surface of elbow. Ulnar nerve found

surrounded by connective tissue in ulnar groove, where it was much thickened, and in forearm where it was glued by scar tissue to inner aspect of olecranon. The nerve was carefully dissected out throughout extent of lesion, and most of the scar tissue was dissected from it. Where nerve was thickened the epineurium was incised to relieve pressure on the fibers. Some bone was chiselled from inner aspect of olecranon and from ulnar groove on internal condyle, and periosteum was carefully sewed over these raw bone surfaces. The nerve was not replaced, however, but was easily moved far from the bone, viz., to the antero-internal aspect of the elbow where it was left, wrapped in Cargile membrane. Skin wound closed without drainage.

March 24—Primary union. Slight motor and sensory improvement. Slight galvanic and faradic response. Much atrophy of ulnar-supplied hand muscles. Thereafter for several months daily galvanic treatment.

1917, January 2—No material improvement. Electric reactions and atrophy of hand muscles as before.

March 10—Evident improvement.

November 6—Atrophy of hand muscles has largely disappeared. Movements of these muscles good. Some paresthesia in hypothenar region.

Upon my return to civilian practice after the war I saw the patient again and made the following note:

1919, June 14—Atrophies in right hand have almost entirely disappeared. Function in affected muscles very good (almost normal). Sensation restored. Still slight paresthesia in hypothenar region, but diminishing.

1921, February 13—Ulnar nerve practically completely restored. There is now to be noted only very slight atrophy of first and fourth dorsal interossei. Nerve trunk at inner aspect of elbow is not thickened nor abnormally sensitive.

In this case the ulnar neuritis began a year or more after the injury and many months after the bone repair. It is therefore an instance of *late* and not merely one of *delayed* ulnar paresis. The report is added to the few above referred to in order to further illustrate, first, that the phenomenon may appear after elbow injuries to adults and, second, that the injury (or inflammation) may be of any type from which a growth of bone or connective tissue may compromise the nerve. (In one of Hunt's three cases of old fracture, a bursal cyst was found pressing on the nerve).

Mouchet recommended, and employed, cuneiform osteotomy of the humerus to release the nerve from stretching and from trauma against the olecranon. Such a procedure would, of course, not be considered in the

absence of a cubitus valgus and even in the presence of such a deformity, neurolysis and transplantation of the nerve to the flexor aspect of the elbow would appear to be more certain of results. The nerve is readily exposed, its liberation requires only a little delicacy, and its dislocation presents no difficulty. Instead of the Cargile membrane used in the case here reported, it is probably better to lightly surround the nerve with fat or with such areolar tissue as is available.

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6. Peltesohn, Siegfried: Zeitschr. f. orthop. Chir., 1906, Vol. 17, p. 246.
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ANNUAL MEETING OF THE AMERICAN ORTHOPEDIC ASSOCIATION.

THE 38th Annual Meeting of the American Orthopedic Association will be held aboard the Chesapeake Steamship Company's Steamship "City of Norfolk," May 15th to 18th, inclusive, 1924.

A Transportation Committee has charge of the arrangements for the boat, and any inquiries should be sent to the Chairman of the Committee, Dr. George E. Bennett, 4 East Madison Street, Baltimore, Md.

The programme of the meeting is as follows:

CLINICAL DAY—THURSDAY, MAY 15, 1924.

9.00 A.M. Operations, Demonstrations, etc., at Johns Hopkins Hospital.
12.30 to 1.30 P.M. Luncheon at Johns Hopkins Hospital.
2.00 to 4.30 P.M. Clinics, Operations, Demonstrations, etc., at the University of Maryland Hospital.
5.00 P.M. Regular session of the A. O. A. on board "City of Norfolk," Chesapeake Steamship Company's steamer. Beginning of cruise on the Chesapeake Bay.
8.00 P.M. First regular session of the A. O. A. on board "City of Norfolk."

FRIDAY, MAY 16, 1924.

9.00 A.M. Morning session of the A. O. A. on board "City of Norfolk."
2.00 P.M. Afternoon session on board "City of Norfolk."
8.00 P.M. Evening session on board "City of Norfolk."

SATURDAY, MAY 17, 1924.

Morning session of A. O. A. on board "City of Norfolk."

The Committee has arranged for the presentation of the following papers:

HIP

"Congenital Dislocation of the Hip—dealing with after-care especially."
Dr. Z. B. Adams

"The Operative Treatment of Congenital Dislocation of the Hip."
Dr. Nathaniel Allison

"Coxa Vara in Adolescence."
Dr. Frederick C. Kidner

"Treatment of Fractures of the Neck of the Femur."
Dr. Frank E. Peckham

SHOULDER

"Two Cases of Chronic Dislocation of the Shoulder."
Dr. W. W. Plummer

"Some Disabilities Following Injuries about the Shoulder with Suggestions as to Treatment."
Dr. James T. Watkins

SPINE

"Sacralized Transverse Process of the Fifth Lumbar—A Method of Removal—A Report of Cases."
Dr. Walter I. BALDWIN

"The Anatomy of the Lumbosacral Region in Relation to Sciatic Pain."
Dr. Murray S. Danforth
Dr. Philip D. Wilson

"Routine Examination of Low Back cases with particular reference to Differential Points between Lumbrosacral and Sacroiliac Regions."
Dr. M. R. Smith-Petersen

"Observations upon an Anatomical Variation of the Lumbosacral Joint, its Diagnosis and Treatment."
Dr. Armitage Whitman

"The Treatment of Tuberculosis of the Spine."
Dr. James O. Wallace

"Subsequent Report on the Treatment of Structural Scoliosis at the Massachusetts General Hospital."
Dr. Armin Klein (by invitation)

INFANTILE PARALYSIS

"End Results of Hoke Operations as done at the Massachusetts General Hospital."
Dr. Lloyd T. Brown

"The Effect of Operations on the Growth of Long Bones in Infantile Paralysis."
Dr. Walter Truslow

MISCELLANEOUS

"Changes in Bone with Alterations in Circulation."
Dr. Sydney M. Cone

"Sutures through Bones and the Attachments of the Tendons to Bones."
Dr. Albert H. Freiberg

"Inferior Radio-Ulnar Dislocation."
Dr. Alexander Gibson

"Bone Grafts."
Major N. T. Kirk, Walter Reed Hospital (by invitation).

"Surgery of Tendons with particular relation to Paralytic and Traumatic Lesions."
Dr. Leo Mayer

"End Results to Illustrate the Value of Fixed Traction in the Treatment of Fractures of the Lower Extremity—100 cases."
Dr. H. Winnett Orr
Dr. James E. M. Thomson

"The Problem of the Orthopaedic Lecture."
Dr. Robert B. Osgood

"A Study of the Reparative Processes of the Body as Evidenced in Clinical Pathology."
Dr. Charles F. Painter

"Fractures of the Os Calcis."
Dr. H. L. Prince

"Skin Flap Methods in Upper Extremity Surgery."
Dr. Arthur Steindler

"The Blood Viscosity in Trophic Disturbances of Circulation."
Dr. Walter G. Stern

"Syphilis of Bones."
Dr. Stewart L. McCurdy

"Synovectomy in Chronic Infectious Arthritis."
Dr. Paul P. Swett

"Pseudohypertrophic Muscular Paralysis."
Dr. Philip Lewin

"Rehabilitation Co-ordinated by Legislation."
Dr. Fred H. Albee

"Rotatory Subluxations of the Knee."
Dr. Charles L. Lowman

The Local Committees for the meeting are as follows:

ENTERTAINMENT COMMITTEE
Dr. Compton Riely, Chairman
Dr. William H. Daniels — Dr. Robert W. Johnson, Jr.

TRANSPORTATION COMMITTEE
Dr. George E. Bennett, Chairman
Dr. J. Albert Key — Dr. I. William Nachlas

BANQUET COMMITTEE
Dr. R. Tunstall Taylor, Chairman
Dr. Sydney M. Cone — Dr. Roades Fayerweather

LOCAL PROGRAMME COMMITTEE
Dr. Robert W. Johnson, Chairman
Dr. Albertus Cotton — Dr. H. L. Wheeler

LADIES' COMMITTEE

Mrs. William S. Baer, Chairman

Mrs. R. Tunstall Taylor	Mrs. Albertus Cotton
Mrs. Compton Riely	Mrs. William H. Daniels
Mrs. Sydney M. Cone	Mrs. Robert W. Johnson, Jr.
Mrs. Roades Fayerweather	Mrs. J. Albert Key
Mrs. George E. Bennett	Mrs. H. L. Wheeler

The Chesapeake Steamship Company has issued the following letter in regard to the trip itinerary:

"To the Members and Guests of the American Orthopedic Association:

"We take this opportunity of advising our members and guests that we have chartered the palatial steamer City of Norfolk for the exclusive use of our party from the time we leave Baltimore until the time we return. We give below the itinerary of this trip:

"THURSDAY, MAY 15, 1924.

Entertainment for the ladies in Baltimore. Automobile excursions and luncheon will be arranged by the Ladies' Committee.

Luncheon at the Johns Hopkins Hospital from 12.30 to 1.30 P.M. for members and guests attending Clinical Day session.

Our party will leave Baltimore from Pier 19, Light Street, at 5.00 P.M.

Entertainment aboard boat. There will be music and dancing, as well as tables for bridge.

We will arrive at Yorktown Friday morning, May 16th.

"FRIDAY, MAY 16, 1924.

We have arranged for an automobile trip for the ladies to leave Yorktown at 10.00 A.M. They will visit "The Cradle of the Nation"—historic Yorktown, Williamsburg and Jamestown. This trip is fully described in the leaflet attached hereto. Luncheon will be served at Jamestown under the mighty oaks that once sheltered Captain John Smith and his faithful band.

The return trip will be make to Yorktown, arriving there about 4.00 P.M.

"SATURDAY, MAY 17, 1924.

Departure will be made from Yorktown sometime during the early morning, arriving at Norfolk about 9.00 A.M., giving an opportunity to view Hampton Roads in the early morning.

Excursion to Virginia Beach. Golf at the Norfolk Country Club or Princess Anne Country Club, at Virginia Beach, for members, guests and ladies in the afternoon.

Sightseeing for the ladies at Norfolk and Old Point Comfort during the morning.

Annual Banquet at 8.00 P.M. at Hotel Monticello, for members, ladies and guests.

"SUNDAY, MAY 18, 1924.

The steamer will leave Norfolk sometime during the early morning, thereby giving a daylight trip up the Chesapeake Bay, arriving at Annapolis about 10.00 A.M.

Golf on the Marine Course, for members, ladies and guests.

Visit to the Naval Academy and historic sites of Annapolis.

Leave Annapolis at 3.00 P.M., arriving in Baltimore at 5.30 P.M., in time to connect with through trains to all points.

Those desiring earlier arrival in Baltimore may leave Annapolis by the electric line every half hour, arriving in Baltimore one hour later, making possible arrival in Baltimore from 11.00 A.M. Sunday.

"The entire cruise on board boat will be within the bounds of the Chesapeake Bay. No seasickness. A folder showing accommodations to be had on board steamer may be obtained from Dr. Bennett.

"The total expense of the entire trip, excluding banquet fee and green fees at the Princess Anne Golf Club, will be $28 per person."

News Notes.

The Section in Orthopedic Surgery of the New York Academy of Medicine met on Friday evening, January 19th.

The following programme was presented:

PRESENTATION OF PATIENTS AND REPORTS OF CASES. (From the Hospital for Ruptured and Crippled.)

(*a*) 1. The infra-articular approach to internal meniscus of the knee joint; demonstration of cases.
2. Synovectomy of knee joint. Percy W. Roberts.

(*b*) Pott's disease complicated by rupture of abscess into bladder. Brainerd H. Whitbeck.

(*c*) Case of plastic surgery of heel. William L. Sneed.

(*d*) 1. Case of hypertrophic villous synovitis of knee.
2. Dislocated internal semilunar cartilage, shown by pneumarthrosis.
3. Case of arthrodesis of right shoulder for obstetrical paralysis. Samuel Kleinberg.

(*e*) Traumatic trochanteric bursitis. Armitage Whitman.

(*f*) Treatment of epiphyseal separation, lower end of radius. Isidor Zadek.

(*g*) 1. Loose body in knee, shown by pneumarthrosis.
2. Case of congenital club feet in adult—operative intervention. Irving Balensweig (*by invitation*).

(*h*) 1. Case of periarterial sympathectomy.
2. Dupuytren's contracture—Gill's operation. Arthur Krida.

At the following meeting, on Friday evening, February 15th, the programme was as follows:

PRESENTATION OF PATIENTS AND REPORTS OF CASES. (From the Hospital for Joint Diseases.)

(*a*) Cases of epiphyseal separation of the upper end of the femur (Frölich's Syndrome). Harry C. Stein (*by invitation*).

(*b*) Lantern slide demonstration of Charcot joints, and joints simulating Charcot joints. Eugene H. Eising.

(*c*) 1. Case of osteomalacia.
2. Case of osteopsathyrosis. Samuel A. Jahss (*by invitation*).

(d) Avulsion of scar tissue in popliteal region treated by moleskin traction. Herman C. Frauenthal.

(*e*) Cases showing results of X-ray therapy in poliomyelitis.
Herman C. Philips,
Walter I. Galland (*by invitation*).

(*f*) Case of progressive myositis ossificans. Harry Finkelstein.

(*g*) Two cases of ostitis of the tibia for diagnosis. Henry W. Frauenthal.

PRESENTATION OF INSTRUMENT:

Simple device for checking drop-foot. Walter L. Galland (*by invitation*).

PAPERS:

(*a*) Radicular pain and its relation to spondylitis deformans from a neurologic viewpoint. (10 minutes.) Charles Rosenheck.

(*b*) "The Ohio state program for the care of crippled children." Albert H. Freiberg, Cincinnati (*by invitation*).

Synopsis: The State of Ohio has evolved a novel plan for adequately treating crippled children. Results have been sufficiently gratifying to warrant its sponsors in urging its adoption by other states. Discussed from the orthopedic surgeon's standpoint.

CORRECTION.

We regret to say that in the January *Journal of Bone and Joint Surgery*, page 164 (A Proposed Modified Fusion Operation on the Spine.—Philip Lewin), the illustration was printed upside down, and also that the Supplementary Note, page 184, should have been placed on page 166, at the end of Dr. Lewin's paper.

Correspondence

Dr. S. L. McCurdy has asked the *Journal* to publish the following letter, a copy of which he has sent to Dr. Lyle.

Pittsburgh, Pa., January 25, 1924.

H. H. M. Lyle, M.D.,
38 E. 68th St.,
New York City.

Dear Doctor Lyle:

It is with regret that I made the error in my article on "The History of Fracture Treatment" which appeared in the *Journal of Bone and Joint Surgery*.

I have in my files a reprint of your article which was printed in the *Annals of Surgery*, June, 1920, and in a manuscript which I wrote regarding apparatus in the treatment of fracture of the femur I may be quoted as follows:

THE SO-CALLED BALKAN FRAME.

> A careful analysis of the Whitney-Juilly frame devised by Dr. H. H. M. Lyle of New York City shows that it was intended for the treatment of compound infected fractures of the femur, that irrigation treatment might be carried out with the greatest degree of care and the least distress to the patient.

Yet, when I was preparing my paper to be read before the Orthopedic Association, I had Blake's reprint before me, which appeared in *Surgery, Gynecology and Obstetrics* (March, 1918, page 245), and entirely forgot what I had previously done upon this subject.

I owe you an apology and am sending the correction to the editor of the *Journal*. The paper which you sent to the editor of the *Journal* was forwarded to me and I am returning it with the hope that the editor will reprint it in its entirety.

Sincerely yours,

S. L. McCurdy.

Current Orthopaedic Literature

FRACTURES AND DISLOCATIONS.

RECURRENT OR HABITUAL DISLOCATION OF THE SHOULDER JOINT. A. S. Blundell Bankart. *British Medical Journal*, December 15, 1923, p. 1132.

This disability may almost be said to be peculiar to athletes and to epileptics. The dislocation is nearly always anterior. It has been thought to be due to abnormal laxness of the capsule, to weakness of the surrounding muscles, and to imperfect healing of an ordinary traumatic dislocation. The various operations for its relief have had as their object the lessening of the size of the capsule, by pleating, or giving it additional support, by transplantation of the muscle. Only those operations which have definitely limited the range of movement of the shoulder joint have succeeded in preventing redislocation, and this probably because of such limitation.

Recurrent dislocation has nothing in common with ordinary traumatic dislocation, which occurs through the anterior and lower portion of the capsule and which heals readily and permanently. It is caused by direct violence against the head of the humerus from behind, causing it to shear off the fibrous capsule of the joint from its attachment to the fibrocartilaginous glenoid ligament. There is no tendency for the detached capsule spontaneously to unite with the fibrocartilage. Hence the dislocation recurs.

The detachment of the capsule from the fibrocartilaginous glenoid ligament is the essential feature in recurrent dislocation.

The author's operation for cure exposes completely the anterior margin of the glenoid cavity through an incision from the upper border of the clavicle above the coracoid, downward and outward for about five inches. The deltoid and pectoralis major are separated and the coracoid defined, divided with the osteotome or bone forceps, and drawn downward with its attached muscles. The tendon of the subscapularis is divided close to its insertion and retracted inward, exposing a rent between the glenoid ligament and the capsule proper. The rent is repaired with interrupted sutures of silkworm gut passed between the free edge of the capsule and the glenoid ligament; the divided subscapularis tendon is reunited, the detached coracoid process reattached, and the wound closed. The arm is kept at rest four weeks and then given active and passive movements. The author reports four cases, with complete restoration of function in three, and with no recurrence in any one of them.—*E. N. Reed, M.D., Los Angeles, Calif.*

FRACTURES IN TRANSPLANTED BONE. S. L. Haas. *Surg., Gyn., and Obst.*, June, 1923, p. 749.

This is a report of a number of experiments on dogs to determine if the cells of transplanted bone possess an independent, inherent power of proliferation and repair

and if the cells remain viable after complete severance of the vascular supply. A metacarpal or metatarsal bone was used in most of the experiments. These were removed, fractured and (1) reimplanted into original position, (2) transplanted into muscles of back in same animal, (3) reimplanted into the muscles after boiling both fragments, (4) transplanted into muscles after boiling both fragments, (5) reimplanted after boiling only one of the fragments, and (6) transplanted into muscles after boiling only one of the fragments.

SUMMARY OF RESULTS.

Fractures in entire free bone transplants, either when reimplanted into original positions or when transplanted into muscles of the back, united as under normal conditions. Fractures of boiled bones, either reimplanted or transplanted, showed no signs of union or proliferation about the ends of fragments. Fractures of transplanted bones, when only one of the fragments was boiled, will unite, whether reimplanted or transplanted into muscles. It is believed that this proves conclusively the independent inherent osteogenetic power of the cells of the transplanted bone.—*R. W. Billington, M.D., Nashville, Tenn.*

CONGENITAL POSTERIOR LUXATION OF THE KNEE. Hartmann-Keppel. *Revue d'orthopédie*, May, 1923, p. 205.

The patient is a man of 44. His mother was of small stature and had double congenital dislocation of the hip. His birth was a difficult breech with considerable traction on the right leg. When he began to walk at 3½, it was noticed that his right knee was abnormal. This condition gradually grew worse until, at present, the right knee is flexed at an angle of 115 degrees and may be pushed back to 70, but cannot be extended. Tne tibia is displaced backward so that the condyles project over it about 5 cm. There is only a vestige of a patella, the quadriceps is undeveloped, and the knee does not function as a joint. The ligaments are so firm that the bones cannot be separated. The femur terminates in only one rounded condyle and the upper end of the tibia is hollowed out obliquely to fit on the posterior aspect of it. Weight is carried on the distal ends of the metatarsals and the toes, the heel never coming down and the tibio-tarsal joint not functioning as such. In fact, the entire tarsal architecture is distorted to accommodate the foot to the equinus position. The musculature of the leg and foot is undeveloped, the psoas acting to carry the entire leg and foot forward in walking.

Aside from the leg deformity, the patient shows a deviation of the nasal septum, Hutchinson teeth, an exaggerated sterno-manubrial angle and knock-knee on the left. He is left-handed. One of his daughters has a congenital dislocation of the hip. This daughter, as well as the patient himself, shows a four plus Wassermann.

The patient would permit no surgical intervention and was content with his deformity. Even if he had consented, it is questionable whether anything could have been done to improve his condition. It would have been necessary not only to reconstruct the knee joint but to remodel the entire ankle and foot. Another method would be to resect the knee and do an astragalectomy.—*William Arthur Clark, M.D., Pasadena, Calif.*

COMPRESSION FRACTURE OF SPINE DEVELOPING DELAYED SYMPTOMS. Fosdick Jones. *Jour. A. M. A.*, Dec. 1, 1923, p. 1860.

Also called post-traumatic spondylitis or Kümmell's disease. Emphasis is placed upon the great importance of a thorough clinical observation in these cases of vertebral injury without cord lesion, together with a complete neurologic and roentgenologic examination. Many of these cases are called back strain, arthritis, contusion, sprains, etc., but develop symptoms and deformity two to six months or longer after the injury.

The pathology is discussed and the literature thoroughly reviewed. The author reports a case in a boy, aged 9 years, with roentgenograms and photographs. It is a well written article and should be read in full to be appreciated. —*F. G. Hodgson, M.D., Atlanta, Ga.*

FRACTURED SPINE: PRACTICAL CARE AND TREATMENT. W. C. G. Kirchner. *Surg., Gyn., and Obst.*, June, 1923, p. 830.

The author states that diagnosis and surgical treatment of this lesion have received much attention recently, but little has been said about many important details in the management of these often difficult and discouraging cases, especially those associated with extensive paralysis. He reports in detail a case of fracture and dislocation of the third and fourth lumbar vertebræ with paralysis of lower limbs and loss of control of bladder and rectum, with complications of decubitus, cellulitis, cystitis, impacted feces, etc. The details of nursing, the uses of plaster shell and cast, hammock suspension of limbs from Balkan frame, surgical care of bed sores, and other practical points are discussed. By extreme patience and care a favorable result was obtained in a hopeless type of case.—*R. W. Billington, M.D., Nashville, Tenn.*

FOUR CASES OF FRACTURE OF THE EXTERNAL HUMERAL CONDYLE. REDUCTION OR RESECTION? H. L. Rocher. *Revue d'orthopédie*, May, 1923, p. 213.

Fracture of the external humeral condyle is particularly frequent in children, constituting 30 per cent of all elbow fractures. It is usually caused by indirect violence, the force being transmitted through the radius. Although the tendency is toward closed reduction of elbow fractures in children, this condylar fracture frequently defies reduction except by open operation. This is especially true for those cases in which the fragment has rotated completely. Of 22 open operations tabulated from reports, 10 were resections of the fragment, 5 simple reduction, 7 osteoplastic. Resection is usually necessary in cases not reduced after a month. The question usually arises whether it is necessary to fasten the fragment with a screw or nail. The author has found that this is not necessary. Suture of the soft parts and position of the elbow holds it. In two cases the position was complete extension gradually flexed after ten days, and in the third, the elbow was immobilized at 75 degrees.

Case 1. Age 8. Fell on the flexed elbow, breaking the external condyle. Fragment displaced outward and backward. Operation five days later. Under the external incision the fragment was found with fractured surface facing outward. Reduction was accomplished without difficulty. The epicondylar muscles were then sutured to the

neighboring firm bone, the elbow was placed in complete extension, and the condyle was thus held in good position. Gradual flexion was begun after ten days. Function of the elbow was normal after four months.

Case 2. Age 12. Direct violence on the flexed elbow. The first surgeon who saw the case found a posterior dislocation and reduced it. Roentgenogram then showed fracture of the external condyle with outward displacement and anterior rotation of 90 degrees. Eight days later lateral incision was made, through which the fractured surface of the condyle presented itself. It was replaced in its normal position and the elbow immobilized in extension. Four months later the elbow showed normal motion, except active flexion which was limited to 80 degrees; passive flexion possible to 20 degrees. Slight outward displacement of the condyle persisted.

Case 3. Age 5. Fracture of external condyle from fall on abducted arm. External displacement and rotation. On opening, the fractured surface was found facing outward. Reduction was not possible until a strand of capsule was removed from between the fragments. Elbow put up at an angle of 75 degrees. After six months the arm was perfectly normal.

Case 4. Age 7. Treated by bandage and massage after fall on the elbow. When first seen by the author a month later function was much impaired. Flexion and extension through an arc of only 20 degrees. Roentgenogram showed fracture of external condyle with displacement outward and upward. At operation the fragment was completely removed and the elbow immobilized in extension. Examination five months after operation showed normal mobility. Roentgenogram showed a filling in of the space where the condyle had been removed.—*William Arthur Clark, M.D., Pasadena, Calif.*

THE ROLE OF THE PERIOSTEUM IN FRACTURES, AMPUTATIONS, ETC. Weinert. *Ztschr. f. orth. Chir.*, 1923, xliv, 1–2 H., p. 100.

After a lengthy discussion with demonstration of pathological specimens, the author ends with the following conclusions: Gun-shot fractures which have been complicated by severe and longstanding infections depend in their consolidation primarily upon the periosteum. In severe cases of war injuries, especially of the leg, with prolonged osteomyelitis complicating the fracture, amputation of the injured limb should not be too long delayed because of the formation of "soft tissue callus" which originates from the periosteum and interferes with a successful result of amputation.—*A. Gottlieb, M.D., Los Angeles, Calif.*

CONGENITAL DEFECTS AND DEFORMITIES.

OPERATIVE TREATMENT OF SPINA BIFIDA. Bade. *Ztschr. f. orth. Chir.*, 1923, xliv 1–2, p. 144.

Author reports a case of a successfully operated spina bifida in a girl of 13. The meningocele sac was immensely large, extending from the 12th dorsal vertebra to the anus and appearing like a rounded sac of 27 cm. in diameter. The spinous processes from the 12th dorsal to the 2nd lumbar were palpable through the sac. The distension of the sac prevented deeper and lower palpation. There were right angle contractures

of both hips, slight contracture of the right knee, and bilateral equino-varus. The skin over and the wall of the sac were so thin that there was danger of rupture. To guard against this and a possible secondary meningitis, it was decided to operate.

Under ether anæsthesia with the patient in abdominal position the operation was performed. Fifteen hundred c.c. of fluid was withdrawn from the sac without ill effects to the patient. After dissecting down to the root of the sac, it was temporarily tied with a bandage to see the effect upon the pulse. No marked changes occurring, the sac was clamped with intestinal clamps, it being so large that two clamps were required. It was stitched with silk suture and covered with a fat muscle flap. Uneventful recovery in two months. The leg and foot deformities were corrected at a later date so that the patient was able to stand and walk with the aid of a walking bench. Until the operation she never could stand up. This case with so large meningocele operated upon successfully seems to be the only one reported.—*A. Gottlieb, M.D., Los Angeles, Calif.*

CLINICAL AND ANATOMIC STUDY OF A CASE OF CONGENITAL GENU RECURVATUM. Boularan and Bounhoure. *Revue d'orthopédie*, May, 1923, p. 245.

At present it is thought that this deformity is a result of compression in the uterus with the knee in bad position. This theory is strengthened by the fact that congenital dislocation of the hip, also supposed to be caused by pressure, often accompanies the knee condition.

The following case is of interest because of the hip dislocation on the same side and because of the anatomic study.

A girl of six and one-half years of age. Family history unimportant. Full term baby; normal delivery. At birth it was noticed that the left knee was markedly curved backward. Nothing was done to correct it at that time. When she began to walk at about three years of age she limped very badly in the left leg. On examination at present the child shows slight adduction, external rotation, and 2.5 cm. shortening of the left leg. Two deep folds of skin anterior to the patella. Slight knock knee, left. Left popliteal space effaced. Left buttock flat, gluteal fold obliterated. Tilting of pelvis to left and compensatory scoliosis. Head of left femur dislocated into the iliac fossa. Flexion of left knee sharply limited, hyperextension exaggerated. Left foot in equinus.

The child walks very poorly, falling frequently. Roentgenograms show the luxation of the hip, elongation of the lower femoral epiphysis, and delayed ossification of the patella. The left patella casts no shadow, while the shadow of the right is present.

Under ether the hip was reduced and immobilized, in flexion-abduction and slight internal rotation. The knee was immobilized flexed to its limit, about 90 degrees. Progress was uneventful for several days, after which the child contracted scarlet fever complicated by broncho-pneumonia and died.

At autopsy the knee was found to be infiltrated with fatty tissue, the muscles pale and fatty. The femoral condyles were narrowed on their horizontal surface and the tibial surface sloped downward and forward. The patella was normal in size and position but entirely cartilaginous. Its external facet occupied about three-fourths of the posterior surface. The crucial ligaments were somewhat elongated and the internal lateral narrow; otherwise the ligaments were normal.

The equinus position of the left foot is explained by the stretching action on the triceps suræ, due to the genu recurvatum.—*William Arthur Clark, M.D., Pasadena, Calif.*

CASE OF CONGENITAL ELEVATION OF THE SHOULDER. A. Contargyris. *La Grèce médicale*, June-July, 1923, p. 34.

Report of a case of a young man, twenty-one years of age, with unilateral congenital elevation of the shoulder, with lack of the rhomboid and lower portion of the trapezius. Pregnancy and delivery normal. The left scapula is 4 cm. higher than the right, and while the upper and internal angle of the left scapula is near the vertebral column, the lower is separated from it. The lower angle of the left scapula is at the fifth intercostal space, while that of the right is at the eighth. There is also a slight scoliosis with convexity toward the left side. In the roentgenogram the left scapula seems smaller than the right, but there is no other abnormality noticed in the rest of the bony skeleton. Right angle abduction of the shoulder. Lack of the rhomboid muscle and of the lower portion of the trapezius, confirmed by electricity.

From the point of view of etiology, the author is of the opinion of Tridon and Delchef, who attribute this abnormality to a lack of descent of the scapula from the first (cervical) position to its definite position behind the thorax between the second and eighth ribs, due to some trouble during pregnancy. The lack of the muscles is probably caused by the same condition.

The patient made a slight improvement under physical treatment, and there was not sufficient difficulty to justify an operation.

CERVICAL RIBS AND HYPERTROPHY OF THE TRANSVERSE PROCESS OF THE SEVENTH CERVICAL VERTEBRA. O. Crouzon. *Presse Médicale*, Nov. 21, 1923, p. 969.

By means of the roentgenogram, hypertrophy of the transverse process of the seventh cervical vertebra is to be distinguished from cervical rib. Of seventy cases with cervical rib syndromes, fifty-six had hypertrophy of transverse processes only and five had cervical ribs and large transverse processes; one had cervical ribs and normal transverse processes, and eight had rudimentary ribs and large transverse processes. In forty-five cases studied clinically, the onset of symptoms occurred at all ages; the condition was more frequent in women; unilateral in twenty-five cases. In only two cases could the cervical rib be palpated. Nearly all had subjective symptoms. In fifteen cases sensory disturbances were present. Motor disturbances were usually limited to weakness. Atrophy was present in ten cases. Circulatory disturbances were present in four cases and pupillary disturbances in eight cases. Eleven patients were operated upon with good results in the majority of cases. The author feels that a spondylitis may bring on the syndrome in asymptomatic cases.—*J. Albert Key, M.D., Baltimore, Md.*

THE OPEN OPERATION FOR CONGENITAL DISLOCATION OF THE HIP. H. P. H. Galloway. *Surg. Gyn., and Obst.*, Nov., 1923, p. 674.

The author quotes Sherman to the effect that of twenty-eight hips which he explored, in only one was the narrow part of the capsule of sufficient width to admit the passage of the head. After reading Sherman's paper he began to use the open operation in selected cases and now uses it exclusively. He now feels that one approaches these cases with confidence and that the treatment is simple, safe, and practically certain to result satisfactorily. His method is as follows: the capsule is exposed by the Smith-Petersen incision, the limb is rotated inward and the capsule

opened by a crucial incision. The limb is then rotated outward, bringing the ligamentum teres into view. This guides the finger into the acetabulum. Nearly always close to the acetabulum a constriction is felt in the capsule. This is divided freely in a downward direction. The head is then manipulated into the acetabulum, often by means of a hip skid, which acts as a shoehorn, to guide the head. With the hip held in the position of stable reduction, the wound is sutured in layers. The capsule is not sutured. A plaster spica is applied. Three weeks later the cast is changed, stitches removed, and weight-bearing encouraged. The casts are changed at six to eight week intervals and at each change the hip is brought nearer to the mid line. In five to six months all dressings are discarded and the child allowed to go about freely.—*J. Albert Key, M.D., Baltimore.*

THE CLINICAL SYNDROME OF PATHOLOGICAL CRANIO-SYNOSTOSES WITH SPECIAL REFERENCE TO OXYCEPHALY. A. Merlini. *Chir. degli Org. d. Movim.*, Oct., 1923.

The etiology of the oxycephaly and, in general, of the entire pathological cranial synostoses is still undefined. Many investigators connect it with hereditary syphilis, while the more recent opinions point to early rickets. This divergence of theories is not necessarily an absolute one, considering that rachitis according to some pathologists is due to syphilitic infection during conception. Bertolotti, upon the basis of clinical and radiologic data, points out the connection between rachitis and cranial synostoses. Meltzer found other rachitic changes present in 85 per cent. of his patients. The author likewise has found more or less numerous rachitic stigmata in all his clinical cases. Among these are the rosary, the thoracic deformity, the bowing of the long bones, the kyphosis, and kypho-scoliosis of the spine, the pathologic conditions of the teeth, as well as gastro-intestinal symptoms, retardation of dentition, tardiness of walking, etc.

The synostosis of the coronary suture was found constantly by the author. The synostosis of the sagittal suture was complete only in one instance, while in others it stopped several centimeters from the lambda suture. There was a venous congestion of the cerebral convolutions localized at the anterior portion of the cranium and along the aforementioned sutures, which constitutes the initial pathologic stage of oxycephaly. These venous congestions and the hyper-vascularization located essentially at the anterior half of the cranium are considered the true cause of the premature synostoses.

Bertolotti observed in his patients in the earliest periods of life, characteristic cranial tabes followed by the development of oxycephaly after the rapid solidification of the skull. The author believes that this venous reticulum superficial to the surface of the cranium is the effect rather than the cause of cranial stenosis. The development of a collateral venous system which through the numerous emissary foramina communicates with the diploic veins is a means of nature by which it overcomes the disproportion between brain and cranial capacity.

Another measure of nature consists in expulsion of the supra-spinal fluid, thereby creating a considerable increase in the capacity of the skull, inasmuch as the intermediate space between cranial wall and cerebral surface represents under normal conditions about 10 per cent. of the total cranial capacity.

A third measure is the displacement of the brain within the cranial cavity, but this is limited by the falx of the dura mater and the tentorium, which both considerably restrict the mobility of the brain.

The fourth measure consists in the displacement of the cerebellum toward the vertebral canal.

These measures to which nature resorts in order to overcome the deficiency of cranial capacity are likely to cause definite clinical symptoms.

In oxycephaly, and in general in cranial stenosis, the inter-cranial hypertension forces the cortex against the laminæ vitrea and creates deeper and more numerous impressions not restricted to the anterior portion alone, but extending over the frontal and parietal bones, and sometimes the entire skull. This hypertension also produces a diminution of the thickness of the diploic layer, bringing both tables closer together, and also produces great alterations in the basal profile which are especially noticeable in the roentgenogram.

There is a diminution in the length of the orbits, sometimes accompanied by exophthalmus, so often seen in the oxycephaly. Other alterations are the prognathism of the superior maxilla; the obliquity in the implantation of the teeth, and especially of the incisors, and the frequent occurrence of adenoid syndromes, held by Bertolotti as characteristic for oxycephaly.

Radiologic Signs: The digital impressions in the skull are clearer and more numerous in younger oxycephalic individuals. They seem to be the first to appear in the morbid picture, and form with considerable rapidity. The alterations in the base of the cranium demand more time in their appearance due to the greater resistance offered by the massive basilary bones.

We know that in the roentgenogram of the hydrocephalic rachitic individual, these impressions are absent and that the anatomic observations of these skulls confirm this radiologic finding. Aside from attributing to the appearance of the digital impressions a considerable diagnostic value, especially due to their very early establishment, the constant disappearance of the coronary and sagittal sutures and the frequent diastasis of the lambdoid sutures are of paramount importance.

Often the radiographic picture of the oxycephalic shows impressions of the venous sinuses, especially the lateral sinuses.

Another feature is the reduction in volume and often entire obliteration of the accessory nasal cavities, the frontal sinus, the ethmoid sinus, the sphenoid sinus, and maxillary sinus, as well as the mastoid cells.

The radiologic examination of the cranial base also shows important alterations in regard to the three cerebral fossæ.

The anterior cerebral fossa presents a greater obliquity, and slopes precipitately towards the median fossa. A constant sign is the vertical position or steepness of the lesser wings of the sphenoid bone.

In order to obtain the sphenoidal angle in the roentgenogram three points are taken: the nasion or union of the nasal-frontal bone; the ephippion or pterygoid tubercle and the basion or anterior border of the foramen occipitale. The value of this angle according to Bertolotti varies from 130 to 135 degrees. In oxycephaly, this angle is increased to 150 to 170 degrees, approximating a straight angle. There is, therefore, a lordosis of the basilary bone instead of the normal kyphosis. It is Bertolotti's merit to have called attention to this feature which he called basilary lordosis. There are also certain changes in the outline of the sella turcica which is contracted in some cases, while in others it appears dilated, of spherical or oblong shape. In many oxycephalics the posterior fossa of the cranium remains unaltered, while in others there may be found a decided deepening, with increase of the antero-posterior diameter, due to the backward

displacement of the occipital bone. These are the cases in which the lambdoid suture remains open, exercising an important vicarious function. The direction of the foramen occipitale in cranial stenosis approaches more the horizontal.

The changes of the facial bones are to a greater extent subordinated to those in the anterior and middle cerebro fossæ. There is a prognathism and hypertrophy of the maxillary bones, dental irregularity, etc.

Symptoms: The symptoms of oxycephaly and especially cranial stenosis vary in number and intensity. Many light or mild cases of oxycephaly are compensated, and aside from the cranial deformity represent no functional symptoms.

Others, however, show very grave pathologic symptoms, often incompatible with life. There is cephalea or headache of the migraine type, and other nervous symptoms such as vertigo, convulsions, epileptic attacks, although these are rather rare and present themselves often in the period following immediately the premature stenosis—that is between the second and fourth years of life, when the brain grows with extraordinary intensity and the optic symptoms consist in bilateral exophthalmus, nystagmus, and loss of vision, which later may go on to complete blindness.

Some of the authors deny the direct influence of cranial hypertension upon the grounds alone that they have not found increased pressure of the cerebro-spinal fluid at lumbar puncture. This objection is not valid because the lesion of the optic nerves arises suddenly after cranio-synostosis at a time when hypertension has not yet been overcome by the various compensatory mechanisms. This hypertension does not persist throughout life, but, as already pointed out, finally becomes compensated.

If at the time of observation the hypertension no longer exists—that is, if it has already become compensated—one cannot from this deny that it has not existed before, and that it has not then in a former period produced the symptoms of optic atrophy.

Among the clinical symptoms, there are also to be mentioned: impairment of the sense of smell and hearing. The latter is not always in connection with lesions of the acoustic nerve, but depends occasionally upon pathologic condition of the middle ear. The eyes are farther distanced than normal and protruding. The nose is aquiline and not infrequently there are polyps of the nose and hypertrophy of the turbinates. The teeth are implanted irregularly, convergent toward the middle line. The hypertrophy of the faucial tonsils and of the lymphatic tissue in the pharynx gives the oxycephalic an adenoid expression.

The influence of the endocrine glands has been studied by a number of investigators. While ample work has been done in investigation of the endocrine glands in different periods of life, the reports regarding the embryonal life are much more scarce. Some authors have found congenital anomalies. Binet refers to concomitance of hair-lip and infantilism. Darier observed hair-lip connected with polydactyly and coloboma of the iris. Bertolotti reports an interesting case of adiposo-genital dystrophy with polydactyly and hypophysis tumor.

Differential Diagnosis: From the standpoint of differential diagnosis, one must distinguish between oxycephaly on one side and hydrocephaly, microcephaly, and dysostosis cleido-cranialis.

In hydrocephaly there is cerebral pressure produced by the increased cerebral-spinal fluid which, due to the persistence of the sutures and of the fontanelles, produces an enormous enlargement of the skull. The hypertension is therefore counterbalanced by the elasticity of the cranial membranous bones. Furthermore, the flattening of the cerebral cortex in hydrocephalus explains the uniform atrophy of the laminæ vitrea and the absence of the digital impressions.

In microcephalus there is thickening of the cranial bones both in the skull and the base; there are no digital impressions and there is also no basilary lordosis. These skulls are very heavy in relation to their size while the oxycephalic skulls are light and slender.

The changes of the cranium in dysostosis cleido-cranialis were thoroughly studied and described by Hultkrantz. This condition is distinguished by an arrest in the development of the bones of the base, especially in transverse direction, manifested by a retraction of the lower portion of the parietal bones. The incomplete ossification of the skeleton of the base is shown here also by the partial persistence of the synchondroses. There are found numerous wormian bones to be persisting.

The facial skeleton in cranial synostosis is reduced in all dimensions. The nasal bones are missing or very small and fused with the frontal bone. The lacrimal bones either do not exist or are united with the maxillary. Other alterations of minor importance, common both to oxycephaly and dysostoses, are the height of the palate, the arrest of development of the accessory nasal sinuses and of the mastoid cells, the irregularity of dentition, and the prognathism.

Treatment: The rational treatment of the cranial hypertension is operative. Guglielmo da Saliceto in the 13th Century was the first to try with the simple means at his disposal the cure of the external hydrocephalus. Modern surgery has devised various daring methods for this condition. Aside from the lumbar puncture there is to be mentioned the puncture of the ventricles; the trephining with drainage (operation of Keen), the permanent drainage of the cerebro-spinal fluid with formation of a valve in the temporal region with cross incision of the dura and outward turning of the four meningeal flaps (Cushing). Drainage of ventricle was also carried out by Krause by means of a gold tube inserted subcutaneously and by Payr by means of a segment of the saphenous vein which he put in one of the ventricles and put into communication with one of the external jugular.

The method of Bramann combines cerebral puncture with palliative trepanation of the cranium. More recently, Schuller of Vienna has described an operation consisting in palliative trephining of the sella turcica by the nasal route.

All these operative procedures, however, up to the present time give only temporary relief and do not succeed in arresting the progress of the disease.

For microcephaly, Fuller in 1878 mentioned the possibility of craniectomy and Lannelongue in 1890 was the first to carry it out. He established artificial sutures destined to favor the expansion of the brain. The intervention, grave in itself, and associated with a high mortality, was only in exceptional cases crowned with success.

Lowenstein in statistics covering 111 cases operated upon in Germany up to 1900 reports 19 immediate deaths; 25 negative results; 10 mediocre results; and 30 cases in which an improvement is claimed without further details.

Gersuny opened the cranium in complete elliptical resection similar to that used in autopsies, after having incised the soft parts by 12 incisions radiating from the vortex over the lateral sides. This operation was carried out in two sittings on account of the gravity of the interference. The results in his case, a baby 12 months old, were so brilliant that the child subsequently was able to devote itself to regular studies.

According to Giocomi, these decompressing operations in microcephalus are irrational. The skull of the microcephalus, he maintains, is too large for the extreme smallness of the brain and therefore is not in need of trephining operations in order to

increase its capacity. Giordino points out that these patients need phosphorus and not trephining.

In oxycephalics conditions exactly opposite to those in microcephalics obtain. Here it is the diminution of the cranial capacity which produces the grave symptoms. As soon as the hypertension appears operation becomes necessary, and it is of importance to operate as soon as possible, say between 3 and 5 years of age, since later the nerve lesions become unalterable.

It is obvious that the methods of Lannelongue using small flaps do not prove sufficient. Lambotte in 1895 offered a method of cranial decompression which might be applied advantageously in oxycephaly.

The case of Lambotte was one of craneo-stenosis with hypertension and the operation aimed at decompression of the brain. He freed the entire cranial vault by complete elliptical incision as in autopsy and similar to the method of Gersuny. The soft parts were incised by eight vertical incisions and the operation carried out in two sittings. In the first, one-half of the cranium was sectioned and after ten days the section of the cranial vault was completed. He noticed that the cerebrum of the brain remained immovable during the operative act and showed no pulsation until the last bone bridge was dissected and the fold detached. This symptom of absence of cerebral pulsation is most important for the diagnosis of hypertension. The child operated upon by Lambotte was three years old. The immediate operative results were excellent. There was a disappearance of the ocular symptoms and return of vision.

This equatorial section and mobilization of the cranial wall is logical and helpful, and the intervention, especially if practiced in two sittings and with proper instruments, is not too grave an interference even in small children. The danger of a long anæsthesia cannot be avoided inasmuch as local anæsthesia is practically out of the question.

Lately, Zorraquin proposed a new method of craniotomy which he calls Universal and which by the considerable extension of the osteoplastic flaps and from their topographical situation might be characterized as frontal-parietal or syncipital craneotomy. The concept of this operation is to obtain a homogenous decompression leaving a large fontanelle in the place of the natural bregma.

A flap is made through the soft parts in form of an Omega with anterior base twelve centimeters long and ten centimeters wide. The nutrition of the soft parts is assured by the large anterior pedicle. The section of the fold is carried out rapidly by a series of trephined holes about three centimeters distant from each other, and completing the cut by means of a circular saw or by Dahlgren's cranitome. Care must be taken not to injure the longitudinal sinus. This craneotomy, less extensive and less traumatizing than that of Lambotte, can be carried out in a single sitting and in a comparatively short time. The author believes that the early diagnosis, to which especially the radiographic findings lend considerable aid, and the craniotomy, not with the limited method of Lannelongue, but with the more radical modification of Zorraquin and Lambotte, will enable the surgeon to obtain unhoped for successes in this group of cranial lesions, which heretofore had been left to themselves until they had produced the most serious functional damages.—*A. Steindler, M.D., Iowa City, Ia.*